

COLUMBIA LIBRARIES OFFSITE
HEALTH SCIENCES STANDARD



HX00042994

RECAP

DISORDERS
OF
RESPIRATION
AND
CIRCULATION

VON NISSEN

RC 39

N 39

Columbia University
in the City of New York

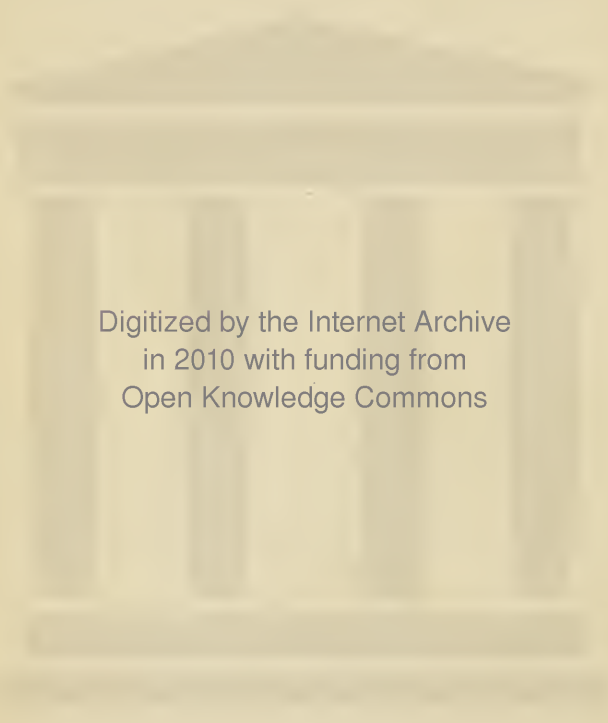
pt. 1

College of Physicians and Surgeons



Given by

Dr. Walter B. James



Digitized by the Internet Archive
in 2010 with funding from
Open Knowledge Commons

DYSPNŒA AND CYANOSIS

DISORDERS OF
Metabolism and Nutrition

A SERIES OF MONOGRAPHS

BY PROF. DR. CARL VON NOORDEN

Professor of the First Medical Clinic, Vienna

I. Obesity	\$.50
II. Nephritis.....	1.00
III. Colitis.....	.50
IV. The Acid Autointoxications.....	.50
V. Saline Therapy.....	.75
VI. Drink Restriction.....	.75
VII. Diabetes Mellitus.....	1.50

Complete Set, **\$5.50.** Also Sold Singly

E. B. TREAT & CO., Publishers, New York

CLINICAL TREATISES
on the
SYMPTOMATOLOGY *and* DIAGNOSIS
of DISORDERS *of*
RESPIRATION *and* CIRCULATION

By PROF. EDMUND VON NEUSSER, M.D.
Professor of the Second Medical Clinic, Vienna; Associate Editor
Nothnagle's Practice of Medicine

AUTHORIZED ENGLISH TRANSLATION

By ANDREW MACFARLANE, M. D.,
Professor of Medical Jurisprudence and Physical Diagnosis,
Albany Medical College; Attending Physician to
St. Peter's and Child's Hospital and
Albany Hospital for Incurables

PART I
DYSPNŒA AND CYANOSIS

NEW YORK
E. B. TREAT & COMPANY
1907

Copyright, 1907
by
E. B. TREAT & CO.,
New York
All rights reserved

PREFACE TO AMERICAN EDITION

THE development of bacteriology since Koch's discovery of the tubercle bacillus in 1881, and the application of solid culture media for the differential growth of bacteria has tended in the last two decades to lead the physician to rely for his diagnoses upon laboratory aids and less upon clinical observation. In order to be thoroughly understood and rationally treated, disease must be studied primarily in its entirety as a pathological process. The physician cannot absolve himself from this responsibility nor find any easy road through the work of another to this desired goal.

The all-absorbing search for the specific cause of a disease, although most valuable when indicated, has too often pushed into the background the manifest clinical evidences of the disease and has regarded them of subordinate value and apparently even of negligible worth.

The diagnosis of disease must in the great majority of patients be determined at the bedside and not in the laboratory. Laboratory findings are most valuable aids to diagnoses, but are not, except in a few instances, diagnoses themselves, and never substitutes for clinical bedside work.

This present series of monographs accentuates the value of the study of symptoms as observed at the

bedside of the patient, and reproduce the marvelous clinical pictures of Trousseau, Niemeyer, Sydenham, Flint and others illuminated by present-day knowledge of pathology and clinical methods.

Prof. Edmund Neusser, with his rare diagnostic instinct and his almost uncanny memory of clinical facts and their co-relation to pathological findings, typifies in the strict sense the modern master clinician.

These lectures are the resultant of an almost limitless clinical material and of a scientific acumen which does not overlook any fact no matter how seemingly trivial and unimportant.

ANDREW MACFARLANE.

CONTENTS

PART I

DYSPNŒA AND CYANOSIS IN DISORDERS OF THE RESPIRATION

CHAPTER	PAGE
I. CONDITIONS WHICH INDUCE DYSPNŒA .	17
Deficiency of Oxygen or Excess of Carbon Dioxid, the Cause of Dyspnœa .	18
Clinical Dyspnœa Accompanied by Cyanosis	20
Absence of Dyspnœa and Cyanosis in Many Disturbances of Respiration .	22
Cyanosis without Dyspnœa	24
II. DYSPNŒA AND CYANOSIS IN DISEASES OF THE RESPIRATORY TRACT	26
Respiratory Tract (Nose and Pharynx) .	26
Laryngeal Affections	27
Diseases of the Trachea	34
Goitre	35
Affections of the Bronchi	37
Bronchial Asthma	40
Pertussis	47
Fibrinous and Capillary Bronchitis . .	48
Pneumonia	51
Hyperemia of the Lungs	55
Edema of the Lungs	56
Pulmonary Emboli and Thrombi . . .	57
Gangrene	61

CHAPTER	PAGE
Pulmonary Emphysema	61
Atrophy and Contraction of the Lungs	66
Pulmonary Syphilis and Actinomycosis .	68
Tumors of the Lung and Bronchi . . .	69
Tuberculosis	71
Pleural Exudates	79
Pneumothorax, Pyo-pneumothorax . .	83
Scoliosis	85

PART II

DYSPNŒA AND CYANOSIS IN DISORDERS OF THE CIRCULATION

I. DYSPNŒA AND CYANOSIS IN CONGENITAL CARDIAC DEFECTS	87
Stenosis of the Pulmonary Artery and Insufficiency of the Tricuspid . .	94
Open Foramen Ovale	94
Open Ductus Arteriosus	95
Stenosis of the Aorta	95
Transposition of the Large Vessels .	96
Mitral Stenosis (Congenital) . . .	97
II. DYSPNŒA AND CYANOSIS IN ACQUIRED CAR- DIAC LESIONS	100
Mitral Insufficiency and Stenosis . .	100
Aortic Lesions	100
Affections of the Pulmonary Valve . .	101
Acute Ulcerative Endocarditis . . .	101
Purulent Aortitis	103
Pericarditis and Adhesive Pericarditis .	104
Hydro-, Hemo,- and Pneumo-Pericardium	106
Fatty and Muscular Degenerations . .	108
Thrombosis of the Heart	110

CHAPTER	PAGE
Theories of Cardiac Dyspnœa (Basch's and Fraenkel's)	III
Final Cause of Cardiac Dyspepsia	113
Dyspnœa and Blood Pressure	115
III. DYSPNŒA AND CYANOSIS IN VASCULAR LESIONS	116
Arteriosclerosis and Anemia	116
Arteriosclerosis and Pseudo-anemia	116
Arteriosclerosis and the Heart	117
Arteriosclerosis and the Kidneys	122
Cerebral and Spinal Arteriosclerosis	124
Significance of Dyspnœa in Arteriosclerosis	124
Aneurism of the Aorta	127
Aneurism and Sclerosis of the Pulmonary Artery	129
IV. DYSPNŒA AND CYANOSIS IN NEUROSES OF THE HEART	131
Cardiac Asthma	131
Paroxysmal Tachycardia	132
V. DYSPNŒA AND CYANOSIS IN DISEASES OF THE GASTRO-INTESTINAL TRACT	134
Diseases of the Stomach	134
Diseases of the Liver and Gall-bladder	134
VI. DYSPNŒA AND CYANOSIS IN INFECTIOUS DISEASES	137
Diphtheria	138
Measles, Scarlet Fever, and Variola	138
Malaria	139
Typhoid, Typhus, Relapsing Fever	141
Erysipelas, Influenza	141
Plague	142

CHAPTER	PAGE
Anthrax	142
Malignant edema	143
Glanders	144
Trichinosis	144
Asiatic Cholera, Peritonitis	145
VII. DYSPNŒA AND CYANOSIS DUE TO POISONS .	148
Morphin, Chloroform, Alcohol, Tobacco	148
Belladonna, Hyoscyamus, Calabar Bean, Physostigmin, Aconite, Colchicum .	149
Strychnin, Camphor, Curare, Conium .	150
Tetroden Fish, Fungus, Agaric Gnat .	151
Barium Salts, Corrosive Substances . .	152
Irritating Gases and Vapors	153
Digitalis, Hellebore and Veratrum . .	154
Phosphorus	154
Carbon Monoxid	154
Substances Forming Methemoglobin . .	156
Antipyrin, Benzol Derivatives, Acetanilid	157
Nitroglycerin, Amyl Nitrit and Carbolic Acid	158
Arseniated Hydrogen and Potassium Chlorat	159
Nitrits—Sodium and Potassium	160
Nitrous Acid, Prussic Acid	162
Sulfuretted Hydrogen	164
Botulism, Tetanus Toxin, Poisonous Fish	165
Insect Stings, Snake Bites	166
Beri-beri, Anesthetic Leprosy	167
VIII. DYSPNŒA AND CYANOSIS IN GENERAL DIS- EASES	169
Bright's Disease	169
Chronic Nephritis	170

CHAPTER	PAGE
Contracted Kidney, Cystic Kidney . . .	170
Renal Asthma versus Cardiac Asthma . . .	171
Latent Nephritis	172
Hemoptysis in Contracted Kidney . . .	173
Cyanosis of Uremia	174
Reduced Hemoglobin in Blood . . .	174
Diabetes	175
Cyanosis in Diabetes	176
Hydrophobia	177
Anemia	179
Leukemia	180
Absence of Cyanosis in Anemia . . .	181
Polycythemia	183
Asthma Dyspepticum	184
Hysteria and Neurasthenia	187
Type of Hysterical Dyspnœa	188
Disturbances of Respiration in Neurasthenia	192
Reflex Asthma	193
Organic Cerebral Lesions	193
Organic Spinal Lesions	194
Neuritis	195
Myositis	198
IX. THERAPY OF DYSPNŒA	200

PART I

DYSPNŒA AND CYANOSIS IN DIS-
ORDERS OF THE RESPIRATION

I

CONDITIONS WHICH INDUCE DYSPNŒA

Limitation in the interchange of gases between the blood and the atmospheric air causes an accumulation of carbon dioxid and a deficiency of oxygen in the blood. Both factors act as irritants to the motor center of respiration, inciting the latter to increased activity. This disturbance when marked gives rise to the subjective sensation of air hunger, or dyspnœa, which is felt the more keenly, the greater the demand for oxygen becomes in the tissue cells. Dyspnœa, therefore, occurs in all cases in which the interchange of gases is obstructed, by depriving the blood of its supply of air, as in laryngeal and tracheal stenosis; by a restricted area of respiration, as in affections of the bronchi and lungs; or by reduced elasticity of the lungs. When pulmonary stasis and diminished power of circulation, as in cardiac insufficiency, interfere with the supply of blood to the respired air in the lungs, or when inability of the blood to absorb the oxygen and transmit it to the tissues, as may be the case in intoxications (carbon monoxid), dyspnœa results.

As is well known, the arterialization of the venous blood by oxydation and decarbonization is effected by

the blood absorbing oxygen from the air and giving up the carbon dioxid. This interchange of gases takes place in accordance with the laws of gas diffusion by equalization of tension. Bohr's assumption that this migration of gases is assisted by a vital reaction or a gas secretion in which, under the influence of the nerves, oxygen is positively sucked up and carbon dioxid expelled, is not sufficiently free from objections to be acceptable without further investigation. It is certainly a tempting theory to look upon the lungs as a gas-secreting gland acting under the influence of the nervous system. The importance of the latter, and especially that of the vagus, would then appear in a new light, so far as the regulation of respiration is concerned.

DEFICIENCY OF OXYGEN OR EXCESS OF CARBON DIOXID, THE CAUSE OF DYSPNŒA

Although it is admitted that dyspnœic respiration is incited by insufficiency of oxygen and excess of carbon dioxid in the blood, it is an open question whether the first or the second is the main factor in producing irritation of the medulla oblongata. Physiologists do not agree as to whether the activity of the respiratory center is regulated by oxygen or carbon dioxid. Pflüger states that marked dyspnœa can arise when oxygen is deficient, even if the quantity of carbon dioxid in the blood is diminished. On the other hand, dyspnœa occurs, even when there is an excess of oxygen, as soon as the carbon dioxid tension exceeds the

normal. Carbon dioxid has been found to undergo in the arterial blood of the woodchuck during hibernation an increase up to 76% without causing dyspnœa. It is conceded, however, that this animal is hyposensitive.

It will be seen, therefore, that an overcharge of the blood with carbon dioxid may be borne very well, and that the influence it exerts upon the regulation of the respiratory function is not yet definitely established. At the same time the diminution of the oxygen tension of the blood as a respiratory irritation is likewise a debatable point. Although the majority of observers attribute the dyspnœa of aëronauts and mountaineers to diminished tension of oxygen and insufficient saturation of the hemoglobin with oxygen, others, for instance Mosso, explain the so-called mountain sickness by a decrease of carbon dioxid in the blood, caused by accelerated evaporation in the rarefied air, which prevents the normal excitation of the respiratory, cardiac and vascular centers.

Deficiency of oxygen and excess of carbon dioxid are therefore both of importance in the causation of dyspnœa. The asphyxia of the newborn, of people working in cellars filled with gas, or of dogs in the *grotta del cane* near Naples, may be laid to both.

Insufficiency of oxygen causes dyspnœa if it occurs suddenly and attains a high degree; gradual diminution of oxygen alone does not cause dyspnœa, as demonstrated by the experiments of Zuntz and Löwy. A slight increase in carbon dioxid, however (even with

excess of oxygen), causes a distinct increase in respirations. Under pathological conditions, at the bedside, both factors—deficiency of oxygen and accumulation of carbon dioxid—are nearly always to be considered, especially in sudden disturbances of respiration, as in acute stenoses of the upper air tract. In slowly developing respiratory difficulties, the accumulation of carbon dioxid in the blood should be held primarily responsible, while deficient oxygen probably plays a subsidiary rôle.

The change in the gaseous composition of the blood is the cause of dyspnœa in the majority of pathological conditions. It may, however, also be due to other causes, either abnormal irritation of the respiratory center, or because the latter is in a state of hyperexcitability. Thus, for example, dyspnœa is found with high temperature, after excessive muscular efforts, in acid intoxications, in organic and functional nervous diseases. In some cases, toxic, easily oxydizable substances may play a part, analogous to the products of fatigue, which accumulate in the tissues in consequence of oxygen deficiency and act as an irritant upon the center of respiration.

CLINICAL DYSPNŒA ACCOMPANIED BY CYANOSIS

At the sickbed dyspnœa is usually accompanied by a second symptom, cyanosis. Formerly cyanosis was regarded as a result of increased carbon dioxid in the blood, but to-day it is recognized that the differences in color between the arterial and venous blood

are not occasioned by the carbon dioxid, but by a changed percentage of oxygen, and that the blue color of the peripheral veins is attributable to interference with light rays in the skin.

Both experimental and clinical observations, especially in intoxications from methemoglobin-forming substances, demonstrate that cyanosis, so far as it depends on the quantity of gas contained in the blood, may occur independently of carbon dioxid saturation. The fact, however, that in the great majority of cases there actually is an excess of carbon dioxid, is explained by the decreased elimination of carbon dioxid from insufficient respiratory function, in disturbances of the respiratory apparatus, and also in affections of the organs of circulation.

Divers, after remaining under water for some minutes, return to the surface in a markedly cyanotic state, occasioned in all probability by both insufficiency of oxygen and excess of carbon dioxid. The primary cause of the cyanosis, however, is the consumption of the circulating oxygen.

The flooding of the organism with oxygenated blood depends upon the quantity of blood circulating in a given unit of time. The rapidity of the circulation of the blood is influenced by the quantity of blood thrown out by the heart into the vessels, and also by the resistance against which the blood is expelled. With diminished motor power and abnormal increase of resistance, the flooding of the organism, and thereby the oxygen supply, must suffer. The diminution of

the *vis a tergo* may, therefore, by retardation of the circulation, lead to consumption of the oxygen transmitted with the arterial blood to the capillaries, and thus cause a reduction to the minimum of the oxygen supply in the venous blood flowing from the tissues. A similar effect may also be produced by an abnormal reduction of the resistance in consequence of dilatation of the abdominal vessels. Owing to the reduction of pressure in the aortic system, the organs do not receive a sufficient supply of blood and become impoverished in oxygen, which gives rise to conditions of collapse, as in peritonitis and partly also in cholera. This accumulated venous blood, deprived of oxygen and enriched with carbon dioxide, causes the cyanotic coloration of vascular and transparent parts of the body. It can attain a high degree of intensity from increased venous pressure, especially from obstructed flow of the venous blood due to insufficient aspiratory action of the right heart. Therefore, not only the chemical factors of diminished arterialization of the pulmonary blood and increased venosity of the capillary blood, but also the mechanical factor of the congested venous system of the general and pulmonic circulations participate in the causation of dyspnoea and cyanosis.

ABSENCE OF DYSPNOEA AND CYANOSIS IN MANY DISTURBANCES OF RESPIRATION

Frequently, however, in extensive pleuritic exudates, incapacitating an entire lung, or in extensive destruction of the pulmonary parenchyma, there is neither

dyspnœa nor cyanosis, provided patients stay quietly in bed. The consumption of oxygen is thus limited to the lowest point and the muscular activity confined to the minimum. Should, however, the need of oxygen be suddenly increased by a diminution of the respiratory surface or by the occurrence, from various causes, of an increased demand for oxygen in the organism, dyspnœic respiration will manifest itself. Dyspnœa, therefore, acts like a regulator, by which the organism defends itself against the impending impoverishment of oxygen. In deeper respirations the pressure of oxygen in the alveoli is increased, with the result that the pulmonary blood absorbs a greater quantity of oxygen. This reinforced respiratory action assists in the evacuation of the blood from the peripheral veins to the heart; the blood stream is accelerated, which also aids in providing the blood with more oxygen and in depleting it of carbon dioxid. It is only after failure to re-establish the normal gas-composition of the blood in spite of assisted aëration of the lungs, that the venosity of the blood increases and cyanosis occurs in consequence of deficient oxygen. In due course the body becomes cold; there is drowsiness; subjective dyspnœa disappears in consequence of insensibility of the brain; respirations become more frequent and superficial, until finally asphyxia sets in, which, by stopping the heart's action, leads to death as soon as the organs are no longer excitable owing to the deficiency of oxygen.

In disturbances of the respiratory apparatus with

dyspnœa, pronounced cyanosis therefore only occurs when the respiration is not strong enough to arterialize the blood through increased pulmonary action. The cause of this may be that the impediment to respiration is too great at the outset, or that the respiratory muscles and their innervation are too weak either from the first or have become weakened in their effort to overcome the obstruction to respiration. For this reason the improvement of a previously existing dyspnœa with the simultaneous appearance or intensification of cyanosis, is an indication of respiratory insufficiency, and, therefore, as a rule, a prognostically ominous manifestation in disturbances of respiration.

CYANOSIS WITHOUT DYSPNŒA

On the other hand, sometimes in deep cyanosis, as for instance in congenital cardiac deficiency, there is an absence of all disturbances of respiration, even with increased muscular work. Owing to the absence of dyspnœa there is no reaction and the supply of oxygen to the tissues and its consumption must be regulated in a different manner. Rapidity of circulation due to compensatory hypertrophy of the heart, increase of the hemoglobin, polycythemia, concentration of the blood by transudation of plasma, may represent the regulating forces employed by the organism to defend itself against the deficiency of oxygen. It is possible also that the organism deteriorates by becoming accustomed to the deficiency of oxygen, and the respiratory center sacrifices its excitability to the increased venosity of the blood.

It is therefore possible that even in extensive respiratory obstruction both dyspnœa and cyanosis may be absent, and that in cases where both symptoms are present they do not always follow a parallel course in that dyspnœa preponderates in disturbances of respiration and cyanosis in those of the circulation. Then, too, there are cases of prolonged dyspnœa without cyanosis. This refers especially to those affections in which disturbance of respiration is due to direct or reflex excitation of the central nervous organs of the respiratory system. It is frequently observed in the dyspnœa with high temperature, in the heavy breathing which precedes coma diabeticum, and in bronchial asthma. In other cases the clinical picture is varied by extreme pallor instead of cyanosis. This may occur in the initial stage of hydrophobia, or in cases where from various causes anemia is present, as in internal hemorrhage, perforating aneurisms, and in penetrating, shooting and stab wounds.

II

DYSPNŒA AND CYANOSIS IN DISEASES OF THE RESPIRATORY TRACT

UPPER RESPIRATORY TRACT (NOSE AND PHARYNX)

In affections of the nose, dyspnœa has been observed to reach such a degree as to assume the character of orthopnœa. The troublesome breathing, during nursing, of the newborn, who suffer from (syphilitic) coryza, is explained by the obstructed nasal ducts. The dyspnœa forces the infant to interrupt the act of nursing, and if this continues sufficiently long, there may be exhaustion and even fatal termination owing to inability to take nutrition. Independently of nursing, dyspnœic manifestations may occur in coryza, severe enough to assume the character of orthopnœa. These paroxysms are explained by the great irritability of the nasal mucous membranes and the prevalence of nasal breathing in the newborn.

In adults with unilateral nasal stenosis, there may be dyspnœic paroxysms at night, especially if patients lie on the side opposite to the stenosis. Lermoyez observed an hysterical girl of fifteen who was unable to breathe through the nose, although the ducts were permeable. Upon covering the mouth with the hand, there was immobilization of the thorax with manifestations of dyspnœa.

Foreign bodies, especially large pieces of badly mas-

ticated food, may stick in the faucial isthmus, causing the danger of suffocation and cyanosis. In this connection a case observed by me is interesting. There was a sudden attack of deep cyanosis and dyspnœa, due to a uvular cyst (*herpes uvulæ*), filled with bloody-serous fluid, the size of a plum and obstructing the faucial isthmus. After the cyst was punctured and emptied, all the threatening manifestations instantly disappeared. Hinsdale described a case of universal urticaria with deep dyspnœa, caused by edematous swelling of the uvula. Evidently the urticaria had become localized in the soft palate, and an application of cocaine soon removed the trouble. Similar swellings have been observed in pemphigus and multiform exudative erythema.

LARYNGEAL AFFECTIONS

Cyanosis with difficulty in breathing is a frequent symptom in affections of the larynx, caused by acute or chronic constrictions or occlusions of the respiratory tract. In laryngeal dyspnœa, the mechanical impediment to breathing is usually complicated by a spasmodic symptom, spasm of the glottis. This spasm occurring in children is sufficient to produce manifestations of suffocation and cyanosis as takes place in tetany, eclampsia and in complications of simple laryngitis, also in inflammations of adenoid vegetations and in the presence of foreign bodies. In these cases both voice and respiration are perfectly normal in the apastic intervals. In adults, laryngospasm, as it occurs in epilepsy, tetany, paramyoclonus,

chorea, hydrophobia and tabes, also in cerebral tumor and hydrocephalus, is, as a rule, less stormy.

In the pseudo-croupous form of catarrhal laryngitis, the spastic element is to be considered in addition to the inflammatory swelling of the mucous membrane. As the spasm relaxes, there are almost entirely free intervals, while in genuine croup the mechanical impediment predominates and the dyspnœa remains permanent, as the spastic element is not an important factor in consequence of the paralysis of the musculature of the glottis. In very severe laryngitis with inflammatory edema below the vocal cords (laryngitis subglottica), difficulty of respiration may persist even in the intervals between the dyspnœic paroxysms. The voice becomes coarse as in pseudo-croup, but not so husky and hoarse as in genuine croup.

Dyspnœa with paroxysms of suffocation and painful oppression, incessant violent irritation to coughing, occurs in measles, partly in consequence of the follicular swelling which accompanies the catarrh in measles, in part as a result of the inflammatory edema of the subglottic zone, and partly from ulceration and erosion of the laryngeal mucous membrane. Sometimes these grave laryngeal symptoms occur in the prodromal stage, and Hebra in his lectures mentioned cases which had been transferred to the surgical division for tracheotomy and in the further course turned out to be suffering from measles. It is a question of prodromal exanthema of the larynx, and as the exanthema appears on the body, dyspnœa rapidly disap-

pears. Cases in which genuine croup with diphtheria bacilli occur as complication of measles must be differentiated. The difficulties of respiration show here a progressive character.

In rare cases varicella may produce manifestations of suffocation if the process becomes located in the larynx. Marfan and Halle have described such cases. The diagnosis may give rise to difficulty in case the larynx is involved before the eruption of the exanthema. In typhoid fever, also, laryngeal symptoms may occupy a prominent place at an early stage, especially in children (laryngo-typhoid).

The usual picture of stenosis of the respiratory tract is well known. The difficulty in breathing occurs especially in inspiration, while expiration, particularly the cough, is suppressed as much as possible. In children suffering from croup the voice becomes hoarse and aphonic, as does the cough. Orthopnœa occurs, and if the attacks are frequent, there is persistent cyanosis. Inspiration is protracted, increased by the auxiliary muscles of inspiration and accompanied by loud stridor. Downward movement of the larynx, inspiratory retraction of the intercostal spaces, fossa jugularis, supraclavicular fossæ and epigastrium, stenotic breath sounds, diminution or entire absence of vesicular respiration, are characteristics of a stenotic attack. Expiration likewise is obstructed in croup, protracted and noisy, and the great effort of all the auxiliary muscles of expiration, which serve to contract the thorax, is evident. The expiratory effort

alone, by which the blood is expressed from the thorax, also the inspiratory insufficiency of aspiration, may cause cyanosis with dilatation of the cervical veins. The effect of croupous dyspnœa is therefore double, as stenosis interferes with both inspiration and expiration.

In most cases of laryngeal stenosis the inspiratory type preponderates. The prototype of this form of dyspnœa is considered to be paralysis of the posterior cricoarytenoid muscles, which may be the only expression of a tabes for some length of time. The difficulty in breathing occurs particularly in great muscular exertions and is accompanied by loud stridor with unobstructed expiration and normal phonation.

Polypi above the glottis usually cause inspiratory dyspnœa, especially when patients take a posture opposite the site of the polypus. The pedunculated tumor obstructs the glottis, while forced inspiration only serves to render the occlusion of the rima still more complete. Polypi below the glottis may obstruct the latter at expiration like a valve, thereby causing expiratory dyspnœa with normal and unimpeded inspiration. Laryngeal polypi develop slowly with progressive disturbances in phonation. Sometimes, however, they may produce sudden attacks of asphyxia which require immediate surgical interference.

Paralysis of the glottic muscles in affections of the pneumogastric nerve, in cerebral hemorrhages, in emboli of the vertebral artery, hydrocephalus, nuclear paralysis of the vago-accessorius, compression of the

oblongata through tumors, pressure on the pneumogastric nerve by contractures, aneurisms of the carotid, metastatic swellings about the neck and jugular foramen, abscesses and tumors, ascending inflammations of the neck from pathological, mediastinal conditions; furthermore degeneration of the pneumogastric nerve as occurs in tabes, amyotrophic lateral sclerosis and in neuritis of the pneumogastric nerve (lead poisoning, alcoholism, beri-beri, typhoid, etc.): all these conditions cause dyspnœa and cyanosis. Similar effects may be produced by myopathic processes of the glottic muscles, as for instance after lues, diphtheria and trichinosis.

Dyspnœa and cyanosis from laryngeal edema is met with in renal and cardiac affections, goitre, amyloidosis of the organs, acute laryngitis—especially its phlegmonous form—perichondritis, especially of the arytenoid cartilages, ulcers of the larynx, syphilis and lepra; also in inflammatory processes of the tongue, palate and tonsils, less often of the parotid, submaxillary glands and the thyroid, in carcinoma of the esophagus and aneurism of the aorta. Laryngeal catarrh which attacks pregnant women or had existed before pregnancy, may lead to serious dyspnœa and, if complicated by laryngeal edema, to death. Dyspnœa in edema of the glottis is chiefly inspiratory, because the aryepiglottic folds close down like valves, and open up again at expiration. This, however, presupposes a certain mobility of the folds. If the infiltration be tense, the dyspnœa will make itself felt in both respiratory

phases. The voice in these conditions is usually little changed, unlike croup, where hoarseness prevails.

Angioneurotic laryngeal edema belongs in this group, also those rare cases which are regarded as urticaria of the larynx; and finally edema occurring from the use of iodine. Marfan and Sevestre record the recurrence of stenotic symptoms after injections of antidiphtheritic serum, a phenomenon similar to the serum exanthema, and caused by the edematous swelling of the laryngeal mucous membrane, and therefore a sequel of the primary affection.

In poisoning with corrosive substances, in scalds, in typhoid, scarlet fever, erysipelas, pyemia, glanders and ulcerative endocarditis, dyspnoea is not a rare concomitant manifestation. In the course of these affections, edema of the glottis may occur owing to ulceration and inflammation of the submucous cellular tissue or to perichondritis.

In typhoid fever, the appearance of dyspnoea with great hoarseness, pain in the larynx, cough, occasionally dysphagia, should even at an early stage of the disease direct attention to ulcerative perichondritis. Also in the later course of typhoid in the fifth and even up to the eighth week laryngeal edema may occur and the stenosis may cause such an intense degree of dyspnoea that tracheotomy is necessary to save the patient's life.

In Ludwig's angina, dyspnoea may be occasioned both by compression of the larynx and by edema of the laryngeal entrance. It may also be due to the phleg-

monous inflammation of the neck extending along the lamellæ of the cervical fascia to the thoracic cavity, which also spreads to the mediastinal and subpleural cellular tissue and penetrates even through the intervertebral spaces into the spinal canal. Dysphagia, interscapular pains radiating anteriorly toward the sternum, and racking pains in the extremities, may appear as concomitant manifestations of the dyspnœa.

Constriction of the laryngeal lumen may also occur through blood effusions from without after traumata and especially if pointed objects, which have been carelessly swallowed, enter the air tract. This may occur in the unconscious or insane, in sufferers from anesthesia of the laryngeal mucous membrane, also in diphtheria, syphilis, tabes, syringomyelia, affections of the pneumogastric nerve and bulbar paralysis, but never according to my experience in hysteria.

The extension of aphthæ into the larynx very rarely produces dyspnœa, and much more rarely still the immigration of ascarides into the larynx.

Dyspnœa, cyanosis, stridor and alteration of the voice, the latter becoming snuffling, quivering and aphonic as in croup, may be due to laryngeal stenosis in retropharyngeal abscesses, which occur for instance in an acute form in young children after an infectious pharyngitis and lymphadenitis, in diphtheria, scarlet fever and erysipelas. The painful dysphagia, the rigid position of the head and neck inclining toward the healthy side, the swelling of the palate, and frequently the painful edema of the lateral cervical region, should

warn the physician to examine the posterior faucial wall in order to detect any fluctuating prominence. With the opening of the abscess, all these ill-boding symptoms disappear instantly, although a short cessation of respiration may occur immediately after the operation, as it does after tracheotomy, but this is not attended with any untoward consequences.

Retropharyngeal abscesses emanating from caries of the cervical vertebræ do not, as a rule, exhibit any such disquieting manifestations; if they produce signs of laryngeal stenosis, the cyanosis is less marked than the anemia attributable to the primary affection. Perilaryngeal abscesses which are frequently untended with hyperemia, edema and fluctuation, may lead to grave dyspnœa and even to death from syncope. Sometimes they develop without symptoms, following a retropharyngeal abscess or after repeated intubations of the larynx.

DISEASES OF THE TRACHEA

In constriction of the lumen of the trachea both dyspnœa and cyanosis occur, as in laryngeal stenosis under similar conditions. The respiration is characterized by whistling râles which reach their climax during inspiration, and the difficulty in breathing is inspiratory, though not so marked as in laryngeal stenosis. The inspiratory downward movement of the larynx does not occur in tracheal stenosis, nor does the backward inclination of the head nor the changes in the voice, which are observed only in paralysis of

the recurrent laryngeal nerve. The deviations from the ordinary type depend upon the site and other conditions of the stenosis; in scars and new formations, for instance, the gravest disturbances in breathing may occur alternately in inspiration and in expiration, or occasionally include both phases of respiration.

Acute tracheal stenosis may be caused by esophageal swellings pressing the posterior wall of the trachea against the anterior. Salvolini described a case in which this swelling was made up of 33 ascarides, clustered together in a bundle.

GOITRE

In goitre, dyspnœa may be occasioned by various causes. Women suffering from goitre may, through swelling of the thyroid, experience considerable dyspnœa during menstruation, which disappears with the menses. In malignant goitre the dyspnœa may be produced not only by compression of the trachea, but also by pressure on the bronchi and metastases in the lungs. In goitre and in Basedow's disease toxic and cardiac factors play a rôle.

The mobile goitre is an interesting phenomenon. It is a very freely movable thyroid, parts of which—though they are usually situated at the normal place in the region of the first tracheal ring—are aspirated sometimes behind the sternum, at times behind the clavicle into the right or left mediastinum, and there produce attacks of asphyxia and disturbances of circulation from pressure upon the trachea and the

large vessels. In some cases there exists abnormal mobility of both trachea and larynx from above downward. This wandering goitre is differentiated by Wölfler from *goître plongeant*, which, situated in the retrosternal or retroclavicular region, is drawn down by inspiration in consequence of its own mobility, and reappears at expiration. These endothoracic goitres may cause the greatest distress, and in certain movements of the head, for instance to the front or back, may lead to sudden attacks of asphyxia, accompanied by cyanosis. An endothoracic goitre is recognized by the immobility of the larynx during deglutition and the participation of a palpable swelling in the upper mediastinum in lateral movements of the thyroid.

It is worth mentioning that by treatment with thyro-iodin a goitre may be made smaller and mobile. If it is displaced behind the sternum, due to the contractions of the sternocleido-mastoid, there may be tension on the pneumogastric nerve, followed by cardiac arrhythmia and even dilatation. Thus it may happen, as seen in one of my own cases, that the administration of thyro-iodin improves the manifestations of tracheal stenosis, but that in consequence of the reduction and migration of the goitre which this treatment produces, grave manifestations may make themselves manifest on the part of the pneumogastric nerve, and the stenotic dyspnoea may give way to a cardiac one. In the case referred to, operative removal of the goitre did away with the tension on the pneumogastric nerve, and after

the operation all cardiac symptoms, arrhythmia, dyspnœa and cyanosis disappeared, at least for a few months.

AFFECTIONS OF THE BRONCHI

In bronchial stenosis, inspiratory dyspnœa occurs with rarefaction of the air, but only in the section of the thorax affected; hence unilateral retraction of the corresponding half of the chest with inspiratory retraction and immobility of the edges of the lungs. In stenosis of high degree there may be immobility of the intercostal spaces, the supraclavicular region and the epigastrium. Auscultation reveals diminished breath sounds or loud dry râles on the side of the stenosis. The cause of the constriction is most frequently pressure from without by tumors of the thyroid, thymus, esophagus, of the mediastinum, the lymphatic glands and the lung, from cold abscesses due to spinal caries, dermoid cysts, also from aortic aneurisms and large pericardial exudates. It may, however, be caused by inflammatory, ulcerative, cicatricial and carcinomatous constrictions of the bronchial wall itself. In one case there was a gummatous infiltration at the point where the left bronchus branches off, accompanied by considerable asthmatic trouble which yielded to an energetic inunction cure.

In cases where foreign bodies are the cause of the stenosis, an important diagnostic sign is furnished by change in the stenotic manifestations and dyspnœa with change in position of the foreign body. Substances taken into the larynx usually produce most violent

manifestations at first, while at subsequent periods there may be not only an abatement of the complaints, but even a remarkable tolerance on the part of the mucous membrane. If the foreign body is situated near the posterior laryngeal wall, edema of the arytenoid region will rapidly occur with hoarseness, dysphagia, irritant cough, bloody expectoration and dyspnoea, while foreign bodies in the recessus pyramidalis usually cause less distress. In addition to the primary changes induced by foreign bodies in the larynx, there may be subsequent disturbances such as local inflammations, edema, decubitus, abscesses, perichondritis and inflammations, which may extend to the mediastinum.

If a foreign body is expelled from the larynx into the trachea, it may be forced back, and after a possible repetition of this excursion finally arrive in the bronchus. During this migration the symptoms, of course, vary with each change of position, violent paroxysms of coughing and suffocation alternating with periods of relative wellbeing. In these cases the dyspnoeic attack shows the expiratory type, and the spastic cough may assume a pertussis-like character. Foreign bodies, however, may remain in the trachea practically without causing distress or symptoms.

An irritating cough with purulent and sometimes bloody expectoration, which narcotics are unable to suppress, and frequently followed by signs of circumscribed pulmonary gangrene and pleuritis, is an important sign in the diagnosis of foreign bodies which

have penetrated into the small bronchi. This is valuable in cases where the anamnesis does not furnish a sufficient clue, as is frequently the case with children. I have myself observed a case where the sputum was even fetid and a circumscribed pulmonary focus with pleuritis was demonstrable. After the patient had expectorated a small piece of chicken bone of whose aspiration he had had no knowledge whatever, all pathological manifestations subsided.

A cardiac cause of dyspnœa may also be present in affections of the respiratory system. Grave disturbances of respiration with cyanosis are met with in many forms of bronchitis, especially in the acute, grave, febrile bronchitis of children, also in senile bronchiolitis, where the grave dyspnœic paroxysms may also be caused by acute dilatation of the heart, a fact which should be borne in mind for the therapy.

There may, however, be dyspnœa in bronchitis, although both ventricles functionate perfectly, and other factors must then be held responsible. Thus, there is considerable dyspnœa, while cyanosis is wholly absent or barely indicated, in those forms of bronchitis, with severe onset, similar to catarrhal forms of bronchial asthma, and characterized by a few fine râles. They cause the most alarming symptoms especially in children, but after a short course, terminate favorably. In these cases the physician may easily be misled into the diagnosis of capillary bronchitis, whose prognosis is very different. A knowledge of the fact that the patient has already suffered from similar

attacks before or that the parents were asthmatic may help to make the correct diagnosis.

BRONCHIAL ASTHMA

The pathological picture of bronchial asthma is composed of neurotic, catarrhal and pulmonary factors, one or another predominating in each case. The asthmatic nature of the dyspnoea is seen in the rapid onset of the attack and the exacerbation of the difficulty in breathing to an alarming extent, almost to suffocation. But the storm abates very soon and the patient returns to his former normal condition. A paroxysm of this kind is recognized either by the unexpectedness of the attack, or by prodromal nervous symptoms, as constriction of the chest, unpleasant sensations in the intestine, at times nasal manifestations such as sneezing or coryza. This is followed by rapidly developing dyspnoea, accompanied by sibilant râles in which expiration especially is aggravated and prolonged, and accomplished only with the aid of all the expiratory muscles, especially the abdominal. Expectoration is scanty and contains large quantities of eosinophile cells, Curschmann's spirals and Charcot's crystals. There is distention of the lungs. The expiratory character of the breathing with protraction of the expiratory phase at the expense of inspiration, and frequently—though not always—retardation of respiration, places its stamp upon this paroxysmal neurosis. Cases in which inspiration is likewise prolonged and the frequency of respiration strikingly increased, rouse the suspicion

of simultaneous complication of the larger bronchi, of the parenchyma of the lungs or of the heart.

In simple, uncomplicated asthma there is no cyanosis, the pulse is of normal tension and frequency and only moderately accelerated. As the affection proceeds, the transitory pulmonary distention develops into a permanent emphysema, and in asthmatic paroxysms both cyanosis and increased frequency of respiration during the attack are often observed. Sometimes blood is mixed with the sputum, and these hemorrhages are the source of the hemosiderin pigment of the alveolar epithelia and leucocytes. It follows, therefore, that the hemosiderin cells, commonly called heart failure cells, are by no means the exclusive diagnostic criterion of cardiac asthma. Similarly, the presence of Curschmann's spirals and of eosinophile cells alone is not sufficient to decide the neuro-asthmatic nature of the affection, as there are also cases of cardiac asthma with spirals and eosinophile cells.

A forty-six-year-old patient for three years prior to his admission into the clinic suffered from asthmatic paroxysms and cough, especially when climbing mountains and doing hard work. The dyspnoëic paroxysms were accompanied by oppression of the chest, râles and profuse perspiration, lasting about a quarter of an hour, and recurring more frequently at night. Later there was edema of the legs and distention of the abdomen. Auscultation during the attack showed diminished respiration with râles and prolonged expiration. Besides, there were the phys-

ical signs of insufficiency of the aortic valves. Blood pressure 115. Upon the administration of digitalis and especially theocin there was abundant diuresis and considerable improvement in the general condition. To all appearances this was a case of cardiac asthma. Sputum during the attacks was rather plentiful, mucous, with muco-purulent flakes, some of which were very tenacious. These portions showed under the microscope dense mucous filaments arranged in spirals, and among them typical Curschmann's spirals with central filament. Aside from numerous pus corpuscles there were a few scattered eosinophile cells and, likewise a great many heart failure cells with partly diffuse, partly clumpy, brownish-yellow pigment. In a few fields there were also numerous erythrocytes arranged in long rows corresponding to the mucous threads. From the clinical point of view we had only a cardiac asthma to deal with, while the microscopic finding of the sputum in addition to the heart failure cells and erythrocytes showed additional elements which belong to bronchial asthma, as eosinophile cells and Curschmann's spirals. Paroxysmal attacks of dyspnoea which are exceedingly similar to bronchial asthma, with sibilant râles and prolonged expiration also occur in chronic influenza, in which, aside from eosinophile cells, there are microscopically and culturally demonstrable small numbers of Pfeiffer's influenza bacillus. Cases of this kind have been observed repeatedly at our clinic; Lord has mentioned such cases and Ortner described several of them. Sputum prep-

arations, stained merely with fuchsin, revealed the presence of the small cocco-bacillus and also an abundant sputum flora, while asthmatic sputum is poor in bacteria. Patients recovering from influenzal infections may likewise present the symptom-complex of asthma, although they had never suffered from this affection. This provocative effect of influenza may show itself in the evanescent stage of the primary affection and then herpes facialis may develop.

A female nurse, twenty-eight years of age, was received at our clinic suffering from asthma with premenstrual exacerbation of the attack, during which the blood pressure was reduced. The sputum contained eosinophile cells, no Charcot's crystals, and was poor in bacteria. A month prior to the occurrence of these attacks she had recovered from influenza. These attacks of asthma following after influenza suggest a toxic-infectious factor, exercising an effect upon the pulmonary terminals of the pneumogastric nerve, the elimination of which may perhaps become possible by suitable antisera. Boucheron reports cases of asthma, which are said to have been cured by antistreptococcus serum.

The clinical picture of bronchial asthma, the sequence of symptoms, the recurrence of paroxysms and the sputum findings, offer sufficient material for differentiation from those forms of a typical angina pectoris, in which there is also expiratory dyspnœa. At the same time there are many cases of bronchial asthma which may offer diagnostic difficulties if they

occur in youthful cardiac patients. This is pre-eminently the case in affections of the aorta which develop on a syphilitic basis. An exact observation of the attacks, the anamnesis, the general impression of the patient, and the success of the selected therapy are alone decisive.

Paroxysmal attacks of dyspnœa in aneurism of the aorta, especially in youthful patients, may likewise give rise to considerable perplexity, particularly when the physician adheres to the asthmatic symptom-complex or to a preconceived notion, and at the same time neglects an X-ray examination. The pronounced character of the expiratory dyspnœa distinguishes bronchial from cardiac asthma and aneurismal asthma, and also from laryngospasm and all conditions of inspiratory dyspnœa, as spasm of the diaphragm and tetany, in which spastically prolonged inspiration passes by a sudden transition into unobstructed expiration. In many forms of bronchial asthma there is inspiratory bulging of the epigastrium and expiratory flattening of the same, which is diagnostically valuable in contradistinction to the tonic spasm of the diaphragm.

Bronchial asthma thus presents a complete pathological picture, and in most cases there is no particular difficulty in the diagnosis, but cases occur in which anatomical lesions of the pneumogastric nerve develop quite a similar appearance, so that one might speak of a pneumogastric asthma. In explaining the mechanism of essential bronchial asthma an exciting process

must be assumed to exist. This lies in the tract of the pneumogastric, exerts its effect on its respiratory, vasomotor fibers and leads to spasm of the bronchial muscles. The catarrhal manifestations are to be looked upon as a sequence of the vaso-dilatation and hyperemia of the bronchial mucous membrane, a secondary factor, which is superadded to the spastic element.

Such cases as a typical bronchial asthma accompanying a carcinoma of the lungs, observed by A. Schmidt, and paroxysms resembling asthma due to irritation of the pneumogastric from pressure of enlarged glands in measles, whooping cough and tuberculosis, observed by Biermer, illustrate the occasional difficulties of diagnosis and warn us not to omit the X-ray examination in cases which are not absolutely clear.

The importance of vasomotor changes in asthma is especially illustrated by cases in which other vasomotor equivalents, such as dermographism, perspiration, tachycardia, hemicrania and angioneurotic edema, occur simultaneously or alternately with asthmatic paroxysms. In these cases a rapid, angioneurotic swelling of the mucous membrane, due to a dilatation of the bronchial vessels, may lead to a diminution of the lumen of the bronchioles and in this way the asthmatic paroxysms without spasm of the bronchioles are explainable. This angioneurotic hyperemia has already been observed by Stoerck and Pieniazek in a tracheoscopic examination of the lower part of the trachea and at the entrance into the bronchi and sug-

gests that the hyperemia may also spread to the bronchial ramifications. In these vasomotor forms of asthma, amylnitrit, will—according to Solis-Cohen—aggravate the intensity of the paroxysms; adrenal preparations, on the other hand, administered internally for a long period, are said to cure the attacks, while the same medication is without benefit in ordinary bronchial asthma.

Thus it will be seen that aside from the mixed forms of bronchial asthma, where all the components—neurotic, bronchitic and pulmonary, act together, there are also cases in which the spastic or angioneurotic form assumes special importance in the pathological picture. These clinical observations are not contradicted by the results of animal experiments, which have demonstrated on the one hand both vaso-constrictors and vaso-dilators in the pneumogastric, and teach on the other hand, according to Sihle's experiments, that peripheral irritation of the pneumogastric nerve can produce a bronchostenosis from muscular spasm. These findings should be duly considered in the selection of the therapy, which in one case consists of vagus poisons, atropin, datura, hyoscyamus, and in other cases attacks the vasodilator element (adrenalin). At the same time the etiological factor should be kept in mind—for instance in urticaria—by avoiding noxious causes or removing possible metabolic anomalies as factors favoring asthma (uric acid diathesis).

The importance of predisposition is especially il-

lustrated by cases in which nervous asthma, like hay asthma, occurs only during pregnancy and so becomes a sign of pregnancy. Whether in these cases asthma is caused by irritation of the pneumogastric or by intoxication is an unsettled question. The prognosis in these cases is serious for both mother and child and may be an indication for an artificial premature delivery.

PERTUSSIS

Paroxysmal dyspnœa, coupled with cyanosis, occurs in the spasmodic coughing of pertussis, but in contradistinction to asthma there is a protracted sibilant inspiration followed by a series of short expiratory puffs. Increased pressure within the thorax produces an engorgement of the venae cavae, and the appearance of dyspnœa and cyanosis in uncomplicated whooping cough is associated only with the attacks of coughing. In complications, however, as marked dilation of the lungs, interlobular emphysema, pneumothorax, or less frequently edema of the glottis, dyspnœa and cyanosis will, as a matter of course, outlast the attacks. Should a febrile affection, an acute exanthema, supervene, the paroxysms of whooping cough grow less in intensity and frequency, and together with them the dyspnœa and cyanosis.

Permanent cyanosis in pertussis with coincident lessening in the attacks of coughing is, as it is after measles, an important diagnostic sign of bronchopneumonia, edema of the bronchial glands, and later tuberculosis of the glands. In these latter cases, dyspnœa

and cyanosis occur from compression of the bronchi, veins and nerves, and it is just in cases where the recurrent laryngeal nerve reacts against the pressure by irritative phenomena, that a wrong diagnosis of relapsed pertussis may be made owing to the pertussis-like paroxysms thereby produced. For this reason dyspnoea and cyanosis with swelling of the cervical veins outlasting the paroxysms demand a careful examination of the larynx and the thoracic organs, including an X-ray examination.

FIBRINOUS AND CAPILLARY BRONCHITIS

Considerable difficulty in breathing, with cyanosis, fever, frothy and sometimes bloody expectoration, is observed in acute fibrinous bronchitis. In chronic febrile, fibrinous bronchitis, cyanosis with violent paroxysms of coughing and at times also hemoptysis, previous to the expectoration of the plugs, are characteristic signs and have a diagnostic importance in cases where simple, subacute, bronchial catarrh undergoes a transition into fibrinous bronchitis.

Chronic, fibrinous bronchitis at times displays an intermittent course, reminding one in the beginning of an attack of bronchial asthma. I observed a case in which, preceding the expectoration of the characteristic clots, the tenacious sputum contained large number of eosinophile cells and Charcot's crystals. In the typical cases of chronic, fibrinous bronchitis, leading to the constriction of the bronchial lumen, the mechanical factor and therefore inspiratory dyspnoea

prevails, while in bronchial asthma the neurotic factor, and with it expiratory dyspnœa, predominates.

The plugs in symptomatic, pseudofibrinous bronchitis, which occurs in ulcerative processes and especially in syphilis of the trachea and bronchi, consist of desquamated layers of the ulcers, whereas the bronchial clots of true fibrinous bronchitis represent secretory products from a relatively unimpaired mucous membrane. Membranous masses in the bronchi may also be developed by tubercular bronchial ulcers and may lead to grave dyspnœa. Fibrinous bronchitis occurs in acute inflammation of the lungs, due to the extension of the alveolar affection to the bronchi, and in these cases dyspnœa is one of the most prominent signs. In other cases, fibrinous bronchitis follows in the wake of laryngeal and tracheal croup. A rare case of membranous bronchitis was observed by Renon in a thirty-nine-year-old woman whose occupation was to sort grain in a moist room. She expectorated whitish green, non-arborescent membranes which consisted exclusively of *aspergillus mycelia* and spores.

In cases where ordinary bronchitis extends considerably there may be early and great difficulty in breathing with cyanosis, so that from a clinical standpoint the designation of suffocative bronchitis is justified. In capillary bronchitis especially, the continuous and progressive dyspnœa with markedly increased frequency of respiration may assume a suffocative character and be fatal from asphyxia by occlusion of the smallest bronchi, due to edema of the mucous membranes or

to the secretion. At the same time there may also be cardiac disorders in acute bronchitis, particularly in influenzal bronchitis, probably in consequence of the toxic effect of the infectious causative factors. These forms of bronchitis may recur in circumscribed foci, accompanied by a great increase in dyspnœa and cyanosis, but according to Fr. Müller they do not directly imply danger to life. The results are different, if the dyspnœa continues progressive, as in bronchiolitis fibrinosa obliterans, which leads to occlusion of the finest bronchioles by connective tissue, and through impeding respiration causes death by suffocation in the shortest possible time. According to Fraenkel the possibility of bronchiolitis obliterans should be borne in mind when these grave symptoms develop in a previously healthy individual whose organs of respiration have been exposed to external, irritating influences, as steam or dust.

In the chronic forms of bronchitis and in long-continued illnesses, dyspnœa develops in consequence of fatty degeneration of the right ventricle of the heart even where the secondary emphysema is of slight extent.

If in bronchitis there is high fever with cyanosis, dyspnœa and great frequency of respiration, it constitutes diagnostically and prognostically an important symptom-complex which may point to the complication of simple bronchitis with broncho-pneumonia.

Focal bronchitis with râles occurs in areas where the visceral pleura is adherent to the costal pleura,

producing atelectasis and broncho-pneumonic foci. Sometimes acute infectious bronchitis may be complicated by the formation of multiple bronchiectases, which add to the pathological picture an entirely new symptom—the intermittent expectoration of large quantities of muco-purulent sputum in paroxysms of suffocation.

In chronic bronchiectasis, dyspnœa and cyanosis mostly depend upon the size of the cavities which may, if complicated with heart disease or emphysema, attain a great size.

Dyspnœa with accelerated superficial respiration and inspiratory retractions of the thorax also occurs in extensive pulmonary atelectasis. The degree of dyspnœa and cyanosis as well as the other signs of the pathological picture depend upon both the original cause of the atelectasis and the concomitant complications.

PNEUMONIA

Dyspnœa in acute pneumonia is of frequent occurrence and is explained by the diminution of the respiratory surface with lessened inspiration in consequence of reduced power of muscular contraction and of pleuritic pains. Dyspnœa, however, does not always exist in direct proportion to the pathological changes in the lungs. It may be very considerable in spite of the slight extent of the pneumonic affection, and again it may be absent although an entire lung is infiltrated. At the time of the crisis, all of the symptoms may recede, although the physical

findings of the lungs disclose no change whatever. It may be assumed that dyspnœa in pneumonia depends not only on the mechanical factor, but also on the toxic influence of the pathological causative factors upon the center of respiration and also on the degree of susceptibility of the latter. Generally speaking, it may be said that dyspnœa out of proportion to the extent of the local changes, especially if the respirations are very frequent and irregular in the absence of local causes, such as pain or hysteria, justifies an unfavorable prognosis.

In simple, uncomplicated pneumonia cyanosis is a late symptom and proportionate to the progressive change in the area of respiration and is most pronounced, therefore, in bilateral pneumonia. In most cases, early cyanosis associated with dyspnœa means involvement of the heart, such as frequently occurs in grave senile pneumonia and in alcoholics. In these cases cyanosis may occur with accelerated respiration and slightly irritating cough, but without any subjective dyspnœa.

Dyspnœa and cyanosis may also be dependent upon the nature of the pathological cause, even if the extent of the pneumonic condition be slight, as in influenzal pneumonia, streptococcal pneumonia, but particularly in the pneumonia of diphtheria, the plague, and glanders, in which not only the toxic influence upon the respiratory center, but also the direct injury to the heart plays a rôle. A peculiar combination of cyanosis and icterus is observable in septic pneumonia, in which

high temperature, loss of consciousness, delirium, coma and accelerated respiration should be regarded as a frequent premortal symptom-complex.

Deep cyanosis may also occur in pneumonia complicated with thyroiditis, mediastinitis, endocarditis, pericarditis, also with malaria with rapid elevation of temperature and in secondary pneumonia in consequence of carbonmonoxid poisoning, in pneumonia resulting from foreign bodies, including deglutition pneumonia as occurs in persons rescued from drowning. Early dyspnœa and cyanosis with abnormally increased frequency of pulse and not excessive temperature has been observed in the so-called asthenic forms of pneumonia, and also in those cases which develop into pulmonary gangrene. In all of these, there are other symptoms to indicate the malignant type of the disease, as enlargement of the spleen and liver, icterus, meteorism, nephritic manifestations, cyanosis, prostration: signs of grave general infection or intoxication. For this reason it should always be considered a prognostically unfavorable sign, if there is early cyanosis in pneumonia and progressively increasing frequency of pulse without corresponding elevation of temperature, particularly if the pneumonic affection is only of slight extent.

Dyspnœa and cyanosis attain a high degree when pneumonia attacks individuals whose cardiac muscle was previously degenerated, as in pre-existing myocarditis, or in obese persons with dilatation of the right ventricle. In these cases the clinical picture

will also disclose other signs of cardiac inefficiency, such as abnormally high frequency of pulse, weak heart sounds, arrhythmia and low blood pressure.

Of prognostic importance are those forms of pneumonia which complicate a pre-existing mitral stenosis, because it admits of the erroneous diagnosis of myocarditis, if the valvular lesion is not betrayed by typical signs and if the arrhythmia with a small, weak pulse stands in the foreground of the symptoms. In these cases marked cyanosis and dyspnoea may undergo improvement under the administration of digitalis.

Dyspnoea and cyanosis may occasionally appear in pneumonia just before the crisis in consequence of dilatation of the right ventricle, if there occurs a backward engorgement of the blood to the right heart from extension of the infiltration and if there are manifestations of edema of the lung. In progressive dilatation of the right ventricle, with accentuation of the second pulmonary sound, cyanosis and manifestations of commencing edema of the lungs, bleeding may be indicated, even in the case of a rapid and weak pulse.

Not infrequently cyanosis is the result of a sudden reduction of the temperature by antipyretics. This is a defebrile symptom which occurs if the cardiac muscles which had been excited by high temperature no longer experience this abnormal stimulation. In the administration of antipyrin and similar febrifuges cyanosis may be especially pronounced.

Sudden cyanosis and dyspnoea with fatal collapse through paralysis of the heart, thrombosis of the pul-

monary veins and edema of the lungs, may follow pneumonia, in convalescent patients who have left the bed too early.

Intense dyspnœa with slight fever occurs in the pneumonia of diabetics. It may take a lightning course, and lead, with manifestations of cyanosis and coma, to a prematurely fatal termination.

In pregnant women pneumonia is a dangerous complication, because both dyspnœa and cyanosis may attain considerable proportions, and the overcharge of the blood with carbon dioxid means a menace to both mother and child. Pneumonia in hysteria may be accompanied by a high degree of dyspnœa standing in no proportion to the extent of the pneumonic affection. This form of pneumonia gives rise to anxiety, but subsides in a normal crisis. Pronounced tachypnœa has an unfavorable prognosis, if it continues throughout a disease in which the respirations rise to sixty, seventy or ninety, but hysteria is an exception. The other hysterical stigmata and the absence of pronounced cyanosis, may in some cases of this description indicate the correct prognosis. In neurasthenics, however, pneumonia as well as other infectious diseases take under certain conditions a mild course and there may even be a favorable influence noticeable upon the nervous symptoms, as, for instance, in asthma.

HYPEREMIA OF THE LUNGS

Dyspnœa and cyanosis may also occur in hyperemia of the lungs. These hyperemias may, especially in the case of old people after prolonged decubitus, lead rapidly or by repeated attacks to death by suffocation.

But here again there are cases where the grave manifestations, even with pronounced unconsciousness, recede, until after a time the same picture repeats itself. In a case I observed, the autopsy disclosed atrophy of the brain. Cutting of the pneumogastric nerve causes hyperemia and edema of the lungs. It appears, therefore, that in the development of pulmonary hyperemia increasing to an edema, not only the mechanical factor of cardiac weakness, but also disturbances of pulmonary innervation in the sense of a vaso-paralysis may play a rôle. This can be observed in cases where hyperemia of the lungs in hemiplegia develops unilaterally, on the side opposite to that of the cerebral lesion.

Gall stones may reflexly lead to hyperemia of the lung, particularly in the right lower lobe, and subsequently to dyspnœa. Where there are also chills and fever, the picture of pneumonia may be simulated. Hyperemia after trauma of the chest and colds, especially in alcoholics, leads not infrequently to edema of the lungs.

EDEMA OF THE LUNGS

Dyspnœa and cyanosis are frequently terminal signs in edema of the lungs, especially in affections of the organs of respiration and circulation. There should be kept in mind embolism of the pulmonary artery, bronchitis, pneumonia, pleuritis, pulmonary tuberculosis, myocarditis and cardiac insufficiency; further, acute exanthemata, typhoid and especially typhus, recurrent fever, influenza, acute articular rheumatism, grave ma-

laria; in rare cases alcoholic poisoning, carbonmonoxid poisoning, snake bites, frost bite and sunstroke; lastly cerebral affections, especially concussion of the brain. Of particular interest is the edema of the lungs occurring in infections due to the bacterium coli as in incarcerated hernia and perityphlitis, in which the bacterium coli is demonstrable in the sputum. Mention should also be made of the edema of the lungs in the aëronaut and that arising *ex vacuo* with albuminous expectoration, which appears chiefly after thoracentesis, especially where there are pericardial adhesions. One of the most frequent causes of acute edema of the lungs is disease of the kidneys, and sometimes pneumonia of very slight extent in nephritic subjects may lead to a rapidly fatal edema of the lungs.

PULMONARY EMBOLI AND THROMBI

The degree of dyspnœa and of cyanosis in emboli of the pulmonary artery depends chiefly on the lumen of the occluded branch. Embolus of the main branch or trunk of the pulmonary artery leads with violent manifestations to sudden death from syncope or to the protracted agony of suffocation. In the former case there is a marked oppression with pallor, in the latter dyspnœa with protrusion of the bulbi, dilatation of the pupils and frequently convulsions.

If the course of pulmonary embolism be protracted, cyanosis may attain a high degree and be accompanied by swelling of the jugular veins, venous pulsation, and sometimes by distinct pulsation in the epigastrium as a consequence of the stormy contractions of the dilated

right ventricle. This is contrary to the asphyctic form of angina pectoris, which, if protracted, sometimes develops cyanosis and dyspnœa, but the cardiac function is, as a rule, abnormally weak and increased function of the right ventricle has been observed only exceptionally in hypertrophic conditions.

In emboli of the medium and small branches of the pulmonary artery, the onset of which is not infrequently accompanied by chill, the sudden dyspnœa and cyanosis may lessen after a short time, only to repeat itself or, after repeated recurrences may lead to a fatal edema of the lungs, to pneumonia, and at times to recovery. The course of such a pulmonary embolus which does not lead to pneumonia and attacks an individual, not suffering from fever, is afebrile. It is just the absence of fever after preceding initial chill with pleurodynia and bloody expectoration, which is valuable for differentiation from pneumonia.

In emboli of the small branches, there are at first fainting attacks, frequently followed by the formation of a hemorrhagic infarct with hemoptysis, but without cyanosis, while multiple emboli sometimes lead to persistent dyspnœa and cyanosis without any stormy onset. Dyspnœa in pulmonary embolus is occasioned partly by exclusion of the corresponding section of the lungs, in part by effusion of blood into the bronchi, partly by reflex irritation of the pneumogastric nerve terminal in the lungs, and, therefore, does not always correspond to the size of the pulmonary focus. In attacks, however, with marked pulmonary hemor-

rhage, dyspnœa is very intense and predominates over the cyanosis.

Emboli of the smallest branches of the pulmonary artery at times do not produce any material symptoms and do not always lead to hemoptysis. But here, also, there may be dyspnœa and cyanosis in consequence of secondary pleural effusions and disseminated broncho-pneumonia.

It is a well known fact that emboli of the pulmonary artery may occur in the course of the most varied pathological conditions. They may follow injuries to the head by the detachment of thrombic masses from the diploic veins, in ileo-cecal and umbilical phlebitis, in thrombosis of the femoral veins, in occlusion of the prostatic veins or pelvic veins, the latter frequently occurring in the puerperium, in tuberculosis, carcinoma, chlorosis, infectious diseases and during convalescence. They may also be produced by massaging a leg having varicose veins and by careless manipulation of the extremities of persons suffering from phlegmasia alba dolens.

In fractures of the long bones, pulmonary emboli occur partly by the detachment of venous thrombi, in part by marrow invading the open veins of the bones. Fat emboli may also occur in phosphorus poisoning, in diabetes, in orthopedic stretching operations on the knee and ankle joints. In the latter case there may be traumatic epiphyseal detachment, and the fat contained in the spongiosa would furnish material for the embolus. In cases of this kind considerable dyspnœa

may be observed with small, frequent and weak pulse, hemoptysis and cyanosis, with complete consciousness until death, especially if the status thymicus prevail as described by Payr. If the fat particles pass through the pulmonary circulation, grave cerebral symptoms may play a prominent part from embolism of the brain. Pulmonary emboli which occur after reposition of incarcerated herniæ and after detachment of dense perityphlitic adhesions, are caused by loosening of the thrombi in the adherent intestinal or peritoneal veins, and their migration through the venous anastomosis between the portal vein and the inferior vena cava.

The most frequent cause of pulmonary emboli is primary coagulations in the right heart, mostly in the right auricle and at the tricuspid valve, in rare cases at the foramen ovale, and in the region of the unclosed ductus arteriosus. Mention should also be made of pulmonary emboli following the entrance of air into veins, as for instance, after extirpation of goitre, in curetting the uterus, and in other surgical operations.

Thrombosis of the pulmonary artery and its branches occurs during convalescence after serious illness, in cachectic individuals, in tuberculosis, or in consequence of compression of the pulmonary artery by tumors and pleuritic exudates. These may lead to severe cyanosis and dyspnœa in the same way as pulmonary emboli, but thrombosis of several branches of the pulmonary artery may at times, if they develop slowly, remain latent without showing any of these symptoms. If they develop in the course of diseases in which there are cyanosis and dyspnœa, they may

occur without materially intensifying these symptoms. Thrombosis during chlorosis, observed in a few cases, is generally unaccompanied by cyanosis. Latent thrombosis in pleuritic exudates may, after evacuation of the fluid, lead to manifestations of a hemorrhagic infarct and subsequently to death from asphyxiation.

GANGRENE

In gangrene of the lungs, cyanosis and dyspnœa may be accompanied by greatly accelerated respiration which depends less on the extent of the gangrene than on the original affection, and the frequent complication with pleuritis and pneumothorax.

PULMONARY EMPHYSEMA

Both dyspnœa and cyanosis are among the well known symptoms of chronic pulmonary emphysema. The patient, when keeping quiet, finds his condition bearable, and dyspnœa occurs only after bodily exertion. In spite of the pronounced dyspnœa, the frequency of respiration is not materially increased, unless there are complications on the part of the lung, bronchi or heart. Dyspnœa is pre-eminently expiratory, with distinct prolongation of the expiration, and the aid of abdominal pressure is needed, especially in bodily exertion. There is deficient expulsion of gas from the pulmonary alveoli during expiration, in consequence of diminished elasticity of the lungs and inspiratory immobility of the diaphragm. There is also insufficient inspiration, which is responsible for the type of high thoracic respiration, produced with the aid

of the inspiratory auxiliary muscles of the shoulder. The deeper the dyspnœa, the more intense are the symptoms on the part of the circulation. The elasticity of the lungs assists in the diastolic dilatation of the heart, and loss of this elasticity must diminish the diastolic filling of the ventricles, causing a stasis in the capillary circulation. This may be responsible for the cyanosis which is particularly noticeable in increased muscular effort.

If in the further course, there is obliteration of areas of capillaries in the lung tissue followed by dilatation of the hypertrophied right ventricle, the intake of oxygen into the lungs is still further obstructed, and the above symptoms increase in intensity. When inefficiency of the right heart becomes established, cyanosis is permanent and is accompanied by swelling of the cervical veins which show little or no change in volume on respiration. Bronchial catarrh favors the development of emphysema and increases the task thrown upon the heart.

There are thus several factors to be considered in the causation of dyspnœa and cyanosis in emphysema. There are, first, the primary changes in the lungs themselves, then the associated bronchial catarrh which not infrequently displays the type of asthmatic bronchitis, and therefore gives rise to considerable distress, and finally secondary degeneration of the cardiac muscle. An unusual event in vesicular emphysema is sudden dyspnœa due to the development of pneumothorax, consequent upon the bursting of an emphysematous vesicle. Many emphysemas are the final stage of bronchial

asthma. Upon all these conditions depends the character of the dyspnœa and cyanosis, especially whether the latter is transitory or permanent.

The preponderance of one of these factors over the others is important for the prognosis and treatment of emphysema. Intensity and frequency of the accompanying bronchial catarrh, and especially the condition of the heart, determine the prognosis. The treatment will differ according to whether the diminished elasticity of the lungs is the primary affection, or whether the bronchitic factor predominates, whether the neuro-asthmatic element is to be taken into account, and finally, whether the cardiac affection, with or without simultaneous arteriosclerosis dominates in the pathologic picture. The pure form of emphysema requires pneumatotherapy, which, on the other hand should be avoided—particularly expiration into rarefied air—if the bronchitic element predominates, the latter requiring rather climatotherapy. The neuro-asthmatic type of emphysema and the cardiac complications also require therapeutic individualization. It can be understood, therefore, that in emphysema, the dyspnœa will be relieved in different ways, according to the preponderance of one or the other of the above factors: one by solanaceous preparations, another by morphin and oxygen, a third by iodine preparations, others by arsenic, still others by alkalies and diet (where gout is a factor), and, finally, in emphysema with cardiac complications, caffein and theobromin preparations and digitalis.

The diagnostic individualization of the purely pul-

monary, the neuro-asthmatic, the bronchitic and the cardiac factors, which is so important for prognosis and therapy, is based partly on physical findings, partly on the examination of sputa. In eosinophile catarrh, a genetic connection with asthma should be borne in mind, and all the symptoms indicating the diagnosis of this affection should be considered. The dry, moist or muco-purulent condition of the catarrh is important for the determination of the bronchitic type. The purely pulmonary form with decreased elasticity of the lungs, and resulting atrophy, is at times determined, aside from the physical findings in the lungs, by the behavior of the cervical veins. The absence of inspiratory collapse in continued cyanosis, shows that the fullness of the veins is the same in both phases of respiration. For the determination of the condition of the heart, all the signs in cardiac diagnosis should be observed, especially the area of dullness, which in severe emphysema is diminished owing to the distended lung encroaching upon the heart, and also from the fact that the left auricle, which materially influences the extent of cardiac dullness, is insufficiently filled in emphysema in consequence of obliteration of pulmonary capillaries and insufficient filling of the pulmonary veins. The cardiac dullness, reduced by the dilatation of the lung, appears still more contracted when compared with cases of emphysema in which there is mitral insufficiency, mitral stenosis or myocarditic dilatation of the left heart. The left auricle becomes dilated and increases the cardiac dullness, so that it is distinctly demonstrable

even in emphysema. In cases of this kind, radiosopic examination may contribute to the understanding of the case.

Insufficient filling of the left heart in extensive emphysema may cause systolic murmurs in the left ventricle, which, with hypertrophy of the right ventricle, may give rise to the mistaken conclusion of a mitral insufficiency complicating the emphysema. On the other hand, a systolic cardiac murmur may be occasioned by secondary relative tricuspid insufficiency, and under these conditions it may also be distinctly heard at the apex.

The deficient filling of the left heart with considerable obliteration of the pulmonary capillaries, of course leads to insufficient nutrition of the aorta and arteries. So long as the right ventricle functionates satisfactorily, this ischemia of the aortic system and the venous engorgement of the general circulation may be compensated, particularly if the patient keeps quiet. If in emphysema, muscular exertion, such as ascending stairs, does not produce noticeable tachycardia and empty arteries, it may be assumed that the right ventricle functionates efficiently, and is capable of performing the increased task. On the contrary, dyspnœa and cyanosis appearing under such circumstances in conjunction with accelerated and small arterial pulse, and especially with signs of congested liver and kidney, would point to inefficient cardiac function.

Dyspnœa is also met with in the so-called interstitial or interlobular form of emphysema, in which the tear-

ing of the pulmonary alveoli allows air to enter into the interlobular and then the mediastinal tissue. In infantile bronchitis and pertussis, or in the presence of foreign bodies in the air tract, interlobular emphysema may by compressing the pulmonary alveoli lead to intense dyspnœa. Emphysema in the mediastinum may occasion severe cyanosis and dyspnœa by pressure upon the large vessels. The appearance of emphysema of the skin in the jugular fossa and at the neck is of diagnostic importance. In mediastinitis postica, due to the swallowing of foreign bodies, as fish bones, there is also emphysema of the neck and face. The dyspnœa, which may be accompanied by fever, dysphagia and pain in the region of the lower neck and shoulders, stands in the foreground of the pathological picture.

ATROPHY AND CONTRACTION OF THE LUNGS

Dyspnœa and cyanosis also occur in atrophy of the lungs especially when complicated with bronchial catarrh, bronchiectasis and degeneration of the muscular substance of the heart. The volume of the lungs is small and the cardiac dullness enlarged only in cases of pronounced atrophy with retraction of the lungs, the result of considerable obliteration of the alveoli and capillaries. An important auscultatory phenomenon in atrophy of the lungs is the peculiar, hissing respiratory sound, similar to puerile breathing. This sharp sound is probably a transmitted respiratory sound coming from the non-atrophied portion of the lung, and in the absence of other catarrhal manifestations

is of importance for the diagnosis of atrophy of the lungs.

Secondary dilatation and hypertrophy of the right ventricle are absent in atrophy of the lungs on account of the marasmus. In synchronous arterio-sclerosis or arterio-sclerotic renal affections, hypertrophy of the left ventricle may occur. The diagnosis may present difficulty in cases where senile atrophy does not attack the entire lung at the same time. There is then partial diminution of lung volume with dilatation of the alveoli and deep position of the diaphragm, presenting upon percussion the signs of emphysema. In these cases it is important to determine atrophy in other organs as the heart or liver, in order to diagnosticate this form of senile emphysema. The demonstration of an atrophied right ventricle is then of special importance. The clinical picture of a pronounced emphysema with low position of the heart presents epigastric pulsation as evidence of the low position of the heart and of the hypertrophy of the right ventricle. If emphysema with low position of the diaphragm can be demonstrated by percussion while epigastric pulsation is absent in all positions of the body, that is in standing, sitting and in ventral posture, and if at the same time serious fatty degeneration of the heart and adhesions to the pericardium can be excluded, then the absence of epigastric pulsation may point to atrophy of the heart and especially of the right ventricle. In pulmonary tuberculosis complicated with emphysema and an atrophic heart, as in ane-

mic tuberculosis, a similar condition may be observed. In these cases we have to deal with a kind of atrophy from inactivity in consequence of the *lessened* quantity of blood and therefore decrease in capacity or there may be direct emaciation of the heart as a part of the general emaciation. In gout, too, I observed this same condition of the heart, namely the absence of epigastric pulsation with demonstrable emphysema. There was atrophy of the liver with stools deficient in bile, no icterus and the diagnosis of atrophy of the heart and liver based on these findings, was confirmed at autopsy.

Dyspnœa and cyanosis are a frequent occurrence in contraction of the lungs, developed in consequence of indurative pneumonia, after influenza or pleuritic exudates, also in the fibroid forms of phthisis, in pulmonary syphilis, anthracosis, chalicosis, siderosis and aspergillosis. In some cases the proliferation of the connective tissue emanates from the bronchial and mediastinal glands which surround the hilus of the lungs. This indurative periadenitis may lead to constriction of the bronchi and the pulmonary vessels, and by extension to the pericardium produce adhesion of the heart to the pericardium, so that in cases of this kind various factors contribute jointly to the production of dyspnœa and cyanosis.

PULMONARY SYPHILIS AND ACTINOMYCOSIS

Pulmonary syphilis occurring in the form of subacute or chronic phthisis or simple chronic induration of the lungs, may lead to dyspnœa and cyanosis. The

dyspnœa may attain a high degree, if there is at same time stenosis of the upper air tract. The picture may be multiform due to diffuse indurations of the lung, the formation of multiple indurative foci of coarse, cicatricial tissue, complications with adherent pleuritis or encysted pleural exudates, so that it may easily be mistaken for malignant tumor, abscess of the lungs or tuberculosis. Dieulafoy specially refers to the marked dyspnœa in pleural exudates accompanying pulmonary syphilis and the continuance of the same after evacuation of the exudate.

In actinomycosis of the lungs, the occurrence of dyspnœa and cyanosis depends partly upon its extent in the lung, in part upon its extension to the pleura, pericardium, the peripleural connective tissue and the thoracic wall. If in the further course of pulmonary actinomycosis the disease extends in different directions and leads to ulceration with perforation of the thoracic wall, there develops as a rule extensive anemia which in part is also occasioned by the amyloid degeneration of the organs. Generally speaking, there is no very severe dyspnœa or cyanosis in the course of actinomycosis, unless there are special localizations of the affection, as, for instance, miliary actinomycosis of the lungs.

TUMORS OF THE LUNGS AND BRONCHI

Severe dyspnœa and cyanosis may occur in malignant tumor of the lungs and bronchi. The dyspnœa is in part due to the localization of the new growth in the lung itself and its metastases, much more fre-

quently, however, to its extension to the mediastinal lymphatic glands. If the larynx becomes compressed or if the tumor extends into the larynx, the dyspnœa may attain a high degree. The clinical picture assumes different forms according to whether pressure is exerted upon the esophagus, pneumogastric, recurrent laryngeal, phrenicus and sympathicus. Dyspnœa with or without wheezing, asthmatic attacks especially at night, paralysis of the vocal cords, dysphagia, vomiting, tachycardia, hiccoughs, hemoptysis, are frequently associated manifestations. In compression and especially in thrombosis of the vena cava, there is cyanosis of the face with marked engorgement of the upper half of the body. The vast majority of patients succumb to the ever increasing dyspnœa, which may even at the onset of the affection—if the pleura is affected—be very severe and as a rule does not subside after evacuation of the exudate. Should the tumors perforate into the large vessels or the auricles of the heart, the manifestations become exceedingly stormy. In carcinoma of the lungs both dyspnœa and cyanosis may, however, also be caused by other and non-carcinomatous affections of the respiratory organs, as pneumonia, pleuritis, or of the organs of circulation, as pericarditis, and especially emboli of the pulmonary artery and thrombosis of the pulmonary veins. Similarly, other malignant and benign tumors of the lung, sarcoma, enchondroma, lipoma, fibroma, osteoma, dermoid cysts and echinococci, may by compression of the air tract and the large vessels as well as by complica-

tions produce cyanosis and dyspnœa. Grave dyspnœa has been observed in emboli of the pulmonary artery with echinococci in consequence of the perforation of a subendocardial colony into the interior of the right ventricle of the heart.

Emboli of the pulmonary artery which have been found in neoplasms of the adrenals are of special importance. New growths of the adrenals may, as is well known, grow into the adrenal vessels, the renal vein and the vena cava superior. These growths may then become detached and cause sudden death by embolism of the pulmonary artery. Such a result happened at an operation for a renal tumor during the anesthesia. The gravest dyspnœa with violent coughing and cyanosis occurs if echinococci break into the lungs.

TUBERCULOSIS

In tuberculosis of the lungs, cyanosis and dyspnœa are usually not common symptoms. In advanced phthisical cases, dyspnœa and cyanosis are frequently absent, although there is present extensive infiltration of the lungs and considerable difficulty in respiration as in pneumothorax. In spite of the diminution of the area for respiration on account of the progressive destruction of the lungs, most phthisical patients do not experience any subjective dyspnœa, although the frequency of respiration increases. The absence of dyspnœa and cyanosis is usually attributable to the autoconsumption of the patients and is explained by the quantitative impoverishment of the blood in red

blood corpuscles, which, by giving up oxygen in the capillaries, produce the characteristic venous blood.

The appearance of intense dyspnœa in phthisical subjects is therefore an accessory phenomenon giving rise to the suspicion that the primary affection has been complicated by some severe disturbance of the circulation or respiration. In some cases it is an acute laryngeal stenosis from perichondritis, or edema of the glottis, and is accompanied by dyspnœa, wheezing and hoarseness. But the respiratory obstruction may lie deeper and be caused by a bronchial stenosis, in consequence of compression by tubercular or ulcerative glands or by a pseudo-membranous bronchitis, as occurs in tubercular bronchial tumors. In other cases the cause is to be found in an acutely developing diffuse bronchitis, in mixed infections with the influenza bacillus and this may be determined by the bacteriological examination of the sputum. In children especially, both dyspnœa and cyanosis may be very intense by the addition of capillary bronchitis. In these cases the possibility of redevelopment of military tubercles should also be kept in mind, especially if in the course of tuberculosis with comparatively slight bronchial manifestations there is considerable cyanosis, marked increase in the frequency of respiration and pulse with unimportant subjective dyspnœa, but with high fever, impairment of consciousness and increasing loss of vitality.

In high fever, the increased difficulty in breathing may also be caused by the fact that the primary

affection is complicated by a pneumococcic infection, in which case the sputum assumes the characteristic properties of pneumonic sputum, and the physical manifestations of pneumonia occupy the more prominent place. In this rusty-colored sputum there are then not only tubercle bacilli demonstrable, but in the beginning of the affection also the diplococcus of pneumonia, which under the microscope appear often as encapsuled cocci. Where the capsule is missing, their virulence is demonstrable on test animals from the pure culture obtained from the sputum. In these cases the dyspnœa can in part be traced to the acute reduction of the area of respiration, and partly also to toxic influences from the mixed infection. This mixed infection, which should be regarded as a diplococcus pneumonia, is characterized by special changes in the blood and by its comparatively acute course. There are also forms of tubercular pneumonia occasioned by reinfection with the tubercle bacillus itself, in which rusty-colored sputa, bronchial breathing and crepitant râles simulate the picture of a fibrinous pneumonia. These forms which A. Fraenkel designates as pneumonic forms of acute pulmonary tuberculosis usually lead to caseation, in rarer cases to extensive induration, bronchial dilatation and indurative adhesions of the pleura. They generally progress with continuous dyspnœa, while cyanosis is less prominent in the pathological picture in consequence of the progressive anemia and the low state of nutrition unless special complications are present,

In mixed streptococcic infections, dyspnœa may be met with, especially in cases where ulcerative pleuritis and pericarditis occur in the course of pulmonary tuberculosis. This is true not only of ulcerations of the peribronchial and mediastinal lymphatic glands, but also of those rare cases of ulcerative peribronchitis which emanate from the smallest branches, extend upward along the lymphatics to the region of the bronchi and lead to ulceration of the bronchi and pulmonary parenchyma, causing death with the picture of acute septicemia. In all these bronchitic, pneumonic and peribronchitic forms of pulmonary tuberculosis, cyanosis and difficulty in breathing may occur in the early and non-anemic cases, when constriction of the bronchi and reduction in the area of respiration acutely interferes with the interchange of pulmonary gases.

In the pleuritic form of pulmonary tuberculosis with insidious development of the exudate the occurrence of great difficulty in breathing and cyanosis is not commonly observed. The absence of these symptoms is explained by the anemia of the patient which is present at the beginning of the pleuritis. If in such cases, dyspnœa nevertheless makes its appearance, it is occasioned either by pain and pleural stitches, especially when the exudates develop rapidly, by the development of exudates in encysted spaces, or by a special localization of the exudates as in diaphragmatic pleuritis.

In adhesions of the pleuræ, the dyspnœa may resemble the picture of asthma or simulate an emphy-

sema of the lungs, if patients are constrained by adhesions and immobilization of the diaphragm to breathe with the upper chest. By overlooking the primary affection, the therapy may be entirely wrong. It is therefore not superfluous to point out that in cases of emphysema or asthma which are not quite clear, the most careful examination of the sputum for the presence of tubercle bacilli may be decisive. The specimen should be prepared by centrifugating a large quantity, and it may also be inoculated into guinea pigs. I know of a case which had been treated for a considerable time as asthma in pneumatic cabinets until a rather profuse hemoptysis resulted. This, together with the history of pleuritis and the finding of the tubercle bacilli in the sputum, which contained no eosinophile cells, revealed the true nature of the affection. It should also be mentioned that there are combinations of pulmonary tuberculosis with bronchial asthma, the tuberculosis developing in patients who formerly suffered or still suffer from typical asthmatic paroxysms. I remember a case where in the sputum not only numerous tubercle bacilli were found, but also eosinophile cells and Charcot's crystals. A stay in the mountains was followed by a considerable aggravation of the condition and an exacerbation of the asthmatic paroxysms. In rare cases asthmatic attacks are observed in tuberculosis immediately after the injection of tuberculin. The sputum in these cases contained eosinophile cells and Charcot's crystals, and the blood too showed eosinophile cells in great abun-

dance. In the sputum of tuberculous women the eosinophile cells sometimes undergo a surprising increase at the time of menstruation. As menstruation in the development of temperature acts similarly to a tuberculin injection, it is possible that both premenstrual dyspnœa and the above findings in the sputum should be regarded from the same standpoint, as due perhaps to a toxic influence.

A not infrequent cause of the sudden development of dyspnœa in pulmonary tuberculosis is pneumothorax, but in the vast majority of cases cyanosis, as well as dyspnœa, does not appear at all or is only slightly indicated, especially when the pneumothorax develops in a circumscribed area, or from a perforation of a severely affected lung incapable of respiration. If, however, dyspnœa exists to an extent to cause anxiety, it should be considered as a prognostically serious symptom. It is caused by a pneumothorax occurring in the less diseased lung while the other lung is severely affected.

In some cases of pulmonary tuberculosis the dyspnœa may be occasioned by glands compressing the pneumogastric nerve, and occurs not infrequently in paroxysmal attacks with violent coughing, defying all treatment. Furthermore, dyspnœa may occur paroxysmally with rapid elevations of temperature, particularly when the nightly exacerbations of fever take place. It is attributable less to the effect of the toxins and rather to the temperature, as after the administration of antipyretics and the reduction of fever by

phenacetin, quinin or hydropathic measures, sponging with vinegar, the dyspnœa promptly subsides.

In afebrile, chronic, pulmonary tuberculosis also, dyspnœa and cyanosis may occur. These forms are designated as *phthisis fibrosa*, and their frequent complications—*emphysema*, pleural adhesions, especially adhesions of the heart to the pericardium—cause the disturbances of respiration to take such a prominent place as to present the affection rather under the picture of cardiac inefficiency. These forms sometimes lead to hypertrophy and dilatation of the right ventricle, and the final phase of the affection may appear under the mask of a tricuspid insufficiency.

In ordinary pulmonary tuberculosis dyspnœa and cyanosis of varying degree may be present in consequence of pericarditis or of changes in the heart itself, as in fatty degeneration of the muscular substance. The pericardial adhesions, which are so frequent in tuberculosis, deserve special attention, because they may, not infrequently, be the cause of a fatal edema of the lungs. I have myself observed a case of tuberculosis complicated by a pleural exudate, where immediately after evacuation of the exudate there was an improvement in the dyspnœa. The patient felt better, but after a few hours a high degree of dyspnœa with albuminous expectoration occurred, leading to a fatal termination with symptoms of pulmonary edema. Cases have been rarely observed, where patients extremely dyspnœic, unconscious, with small and often irregular pulse, presenting a moribund appearance, re-

covered rapidly and completely, only to succumb to a fresh recurrence of the attack. The autopsy in these cases revealed tuberculosis of the heart muscle.

One of the more frequent causes of grave and suddenly developing dyspnœa and cyanosis in tuberculosis is embolus of the pulmonary artery from thrombosis of the peripheral veins. Dyspnœa and cyanosis also occur in those forms of pulmonary tuberculosis which develop secondarily in a pre-existing cardiac insufficiency, as congenital mitral stenosis or affections of the pulmonary valves, and especially in aneurisms of the thoracic aorta. This is also true of tuberculosis in kyphoscoliosis where the tuberculosis develops in those sections of the lung which are poorly nourished owing to the deformity. In caries in the region of the fourth cervical vertebra severe dyspnœa may occur when the affection spreads to the nucleus of the phrenicus and is followed by paralysis of the diaphragm.

Intense dyspnœa with violent paroxysms of suffocation may occur in tuberculosis through the erosion of small aneurisms or of large blood vessels into cavities. Cases where ulcerative tuberculous glands break through into the pericardium or aorta may be mentioned as rarities.

Furthermore, dyspnœa is found when tuberculosis is complicated with nephritis, especially in cases where the blood pressure is raised. As is well known, most cases of advanced tuberculosis have, as a rule, a low blood pressure, but where there is coincident nephritis, this rule usually—but not always—shows an excep-

tion, and this refers especially to cases which have a pronounced tendency to dyspnœa and chronic edema of the lungs.

Finally there is the dyspnœa of tuberculosis complicated with hysteria or neurasthenia. A tuberculous female patient in our clinic had paroxysms of dyspnœa at times during the day, especially when she knew she was being observed, and these paroxysms bore the precise character of hysterical tachypnœa. To this group also belong those forms of paroxysmal dyspnœa experienced most frequently at night in neurotic and neurasthenic patients. These cases have been cited by See and A. Fraenkel as examples of so-called pseudo-asthma.

PLEURAL EXUDATES

In pleural exudates, dyspnœa occurs coupled with cyanosis. These phenomena depend less on the size of the exudate than on the rapidity with which it develops, partly also on the nature of the causative factor and on the concomitant and original pathological conditions. There are cases of exudative pleuritis which are ushered in with repeated chills, at times accompanied by high fever and sometimes by expectoration of bloody sputum. This stormy introduction characterizes especially suppurative pleuritis and the exudate may develop with such rapidity as to necessitate its evacuation within a few days. These forms simulate the picture of pleuro-pneumonia and may give rise to a wrong diagnosis of genuine pneumonia.

Dyspnœa and especially cyanosis have considerable diagnostic significance, as the latter is usually a late symptom in genuine, uncomplicated pneumonia. In very large exudates there is deep cyanosis of the whole body in consequence of the severe disturbances of circulation due to the limitation of the thoracic space, and there is besides edema of the ankles and later general dropsy. Marked dyspnœa and cyanosis also occur in comparatively small exudates, if they are mediastinal, and when there is also pronounced interlobular empyema.

Intrathoracic carcinoma and sarcoma causing effusion into the pleural space may, as mentioned before, produce or increase cyanosis and dyspnœa by enlargement of the glands and compression of the vena cava, of the pulmonary artery and veins, or by the direct extension of the growth to the walls of the vessels, with resulting thrombosis and embolism. Such thrombi in the branches of the pulmonary veins may be carried into the general circulation, especially on evacuation of the exudate. In other cases dyspnœa is coupled with anemia and especially in malignant neoplasms with bloody effusions in the pleural cavity. The persistence of dyspnœa or only its slight relief after evacuation of the effusion from the pleural cavity in the case of a carcinoma has already been mentioned.

Cyanosis and dyspnœa in pleuritis may, aside from the quantity of the exudate, attain a high degree owing to the accompanying paralysis of the diaphragm, especially in pleuritis diaphragmatica, where violent

diaphragmatic pains and involvement of the pericardium play a part.

In combination with concomitant or pre-existing affections of the heart and kidney, dyspnœa may be intensified to orthopnœa. The patient has to sit up in bed in order to remove from the lungs the exudate accumulated at the base of the lungs.

The gravest form of dyspnœa may occur from perforation of the pleural space by echinococci. The dyspnœa constantly and at times rapidly increases, and is generally the prominent symptom of intrathoracic echinococci. Death from suffocation is not a rare occurrence in contradistinction to pleuritic exudates. Test punctures of the pleural and pulmonary echinococci may be followed, in addition to urticaria, by grave conditions of collapse. There may be death from suffocation if the sac should rupture and the contents of the cysts should occlude the respiratory tract.

In contradistinction to this grave dyspnœa and cyanosis, after effusion and perforation into the pleura, there are numerous cases, not only of moderate but also of large exudates, in which there is complete absence of dyspnœa or cyanosis and especially if there is no fever and the patient lies quietly in bed. The least physical exertion, sitting up or rising, may produce considerable difficulty in breathing and cyanosis. The cause of these attacks varies: kinking of the trachea or of the vena cava; weakness or thrombosis of the heart; pulmonary edema; perforation of an

exudate into the lungs, mediastinum or pericardium. Difficulty in breathing may also occur in slight exudates, if the pleuritis develops in encysted spaces, walled off by former adhesions. The dyspnoea, which not infrequently is associated with pains in the affected side of the chest, is caused by the impediment to respiration and circulation, formed by the adhesions and complicated by the space-restricting pleuritic effusion.

Similarly, cyanosis and dyspnoea occur in total adhesion of the pleural layers. As the adherent lung cannot expand in its vertical diameter, the thorax is forced to undergo more than normal expansion in the frontal and sagittal diameters, and hence the costal type of respiration as it exists in emphysema of the lungs. The adhesion of the pleural layers follows pleuritis in the course of chronic tuberculosis. The pleuritis is not infrequently only a part manifestation of a general serositis, and in consequence of simultaneous adhesions of the pericardial layers, of the diaphragm and liver, the clinical picture may have multiform features. The dilatation of the thorax in pleural adhesions recalls that of emphysema. The protracted expiration, the asthmatic paroxysms and the hypertrophy of the right ventricle are apt to cause difficulty in the differential diagnosis of both affections. Secondary bronchial catarrh develops in the poorly nourished portions of the lung, and like emphysema, may lead to dilatation of the right ventricle, which may become distended enough to cause a relative tricuspid insuf-

ficiency. Both cyanosis and dyspnœa may then attain a very intense degree, especially if there is simultaneous adhesion of the pericardium to the heart and weakness of the heart, especially of the right ventricle.

PNEUMOTHORAX, PYO-PNEUMOTHORAX

Pneumothorax leads to dyspnœa and cyanosis only if the collapsed lung was not previously materially affected or in good functioning condition, as happens in stabbing and shooting wounds and in emphysema. The lung not only of the affected, but also of the opposite side becomes retracted, from the entrance of air into the thoracic space. The mediastinum is then displaced if it be pliable and unaffected. If it contains indurative tissue and if the lung on the side of the pneumothorax is partially adherent to the thoracic wall, then the healthy side will be less impeded in its movements than when the respiratory organs are relatively healthy. On these factors then: the condition of the lung on the side of the pneumothorax, the behavior of the mediastinum and of the opposite lung, depends the occurrence of dyspnœa in pneumothorax.

When speaking about dyspnœa in tuberculosis, reference was made to the fact that dyspnœa is absent or only faintly indicated when the pneumothorax is circumscribed or has followed the perforation of a seriously affected lung adherent to the pleura and therefore useless for purpose of respiration. If, on the contrary, pneumothorax occurs on the side of the less

affected lung with a seriously affected lung opposite, there may be the most intense dyspnœa leading to sudden death from asphyxiation. These sudden deaths in pneumothorax occur in cases where the pulmonary changes are not marked or where there is a quiescence in their further development. They are fully explained by the mechanism of pneumothorax and the rapid functional collapse of the lungs. The condition of the pulmonary fistula may also come into question. In the case of a closed or valvular pneumothorax the retraction, and therefore also the dyspnœa, may attain an intense degree in consequence of the high positive pressure in the pleural cavity, while in an open pneumothorax, unless it is bilateral as may be the case in penetrating chest wounds, dyspnœa may—*ceteris paribus*—not be present at all or only moderately, and the want of oxygen be supplied by more rapid and deeper respiration. In simultaneous effusion of fluid, that is in pyo-pneumothorax, the increase of the exudate and its character is of some, but not of much importance.

In penetrating shot wounds of the chest which open the pleural cavity dyspnœa and cyanosis are common manifestations. In shots entering the lungs hemoptysis is a constant symptom in addition to those of pneumothorax. An interlobular and mediastinal emphysema increases the dyspnœa, and may, if its onset is rapid, lead to death from suffocation. While dyspnœa and hemoptysis constitute the most important signs of injuries to the pleura and lungs, cyanosis may

be completely absent, and anemia—in addition to the dyspnœa—may prevail in cases where an important vessel has been injured. As the hemorrhage continues, the patient becomes more and more anemic, the pulse smaller, the respiration superficial, and in this condition death often occurs from syncope.

SCOLIOSIS

Dyspnœa in scoliosis may be caused in different ways. It may be occasioned by pressure, atrophy of the lungs and the impossibility of distending the thorax. This results in a compensatory acceleration of respiration attended by almost exclusively abdominal breathing as long as the right ventricle functions sufficiently. If the latter fails, the deficient distention and contraction of the pulmonary alveoli may cause insufficient circulation in the lung and interchange of gases, and dyspnœa is the result of the oxygen famine. This dyspnœa is based on the diminished quantity of blood transmitted into the circulation and materially differs from the dyspnœa of engorgement where the lungs are overfilled with blood, which flows under high pressure into the dilated left auricle. The former receives less blood from the pulmonary veins, and thus both the left auricle and the left ventricle are frequently found small and atrophic.

This condition, however, is by no means the rule in scoliosis. There are cases of kyphoscoliosis with cyanosis, enlargement of the liver and dropsy from venous

obstruction, in which the absence of dyspnœa is a surprising contradiction. These patients can lie on their backs or even sit up in bed without experiencing distress or dyspnœa. This reminds one of cases with congenital insufficiency of cardiac function. I have seen a patient suffering from kyphoscoliosis with considerable edema of the lower extremities, who displayed this surprising contradiction of symptoms as long as his pulse was good and the action of the left ventricle sufficient. To explain the cyanosis in these cases, it is necessary to assume that the right ventricle is comparatively weak, and as the left ventricle is strong, no dyspnœa develops, but only symptoms of passive congestion in the general circulation. If, however, the left ventricle, too, becomes inefficient, if the pulse is weak, frequent and irregular, then there is added to the passive congestion of the circulation an engorgement of the pulmonic circulation which finds expression in dyspnœa with asthmatic attacks. Leyden has already pointed to the functional differences between the right and left ventricles in his work on the fatty heart. I lay special stress upon these cases, because they are of interest from both a prognostic and therapeutic point of view. The point to which treatment is to be directed is not improvement of cardiac function, but relief of the work performed by the general circulation, by deflection to the intestines, by diuresis or venesection. It is a struggle between the right ventricle and the diminished area of respiration in the pulmonic circulation, while the left ventricle is

involved, *only* as it may be expected to perform increased work owing to the engorgement of the venous capillaries. If, however, the left ventricle also becomes exhausted, there will be a dyspnoea similar to cardiac asthma, to which more explicit reference will be made later on.

PART II

DYSPNŒA AND CYANOSIS IN DIS-
ORDERS OF THE CIRCULATION

I

DYSPNŒA AND CYANOSIS IN CONGENITAL CARDIAC LESIONS

In the beginning it was explained that in affections of the respiratory system dyspnœa was a frequent manifestation, and that the associated cyanosis was, in the great majority of cases, attributable to complications from cardiac insufficiency, or else to grave disturbances in the interchange of gases in the lungs. In affections of the circulation, the majority of cases of dyspnœa are complicated by more or less pronounced cyanosis of all degrees of intensity, from pure blue to blue black.

The most marked grades of general cyanosis occur in congenital cardiac insufficiency, and as a rule exist from birth or earliest infancy. In acquired cardiac defects cyanosis never attains so high a degree and runs to a certain extent a course parallel to the intensity of the subjective symptoms, especially the dyspnœa. It is usually a late symptom and generally accompanies the dropsy. Congenital cardiac defects sometimes show a striking contrast between the extent of cyanosis and the subjective symptoms, especially the dyspnœa. Persons suffering from the most intense cyanosis may be able to perform heavy work with-

out loss of breath. These are, however, exceptional cases, and usually cyanosis is accompanied by dyspnoea which may be severe enough to threaten suffocation. Nevertheless, cyanosis in congenital cardiac defects, whether it has existed from earliest infancy or has gradually increased, occupies the foreground of the clinical picture. It is also surprising that in many congenital forms of cyanosis the small peripheral veins are dilated to the utmost, while the large veins show no distention, and there is frequently a disproportion between the cutaneous cyanosis and the stasis in the internal organs. In acquired cardiac defects edema precedes severe cyanosis, or at least accompanies it, while the opposite holds good for congenital lesions, and edema occurs late or not at all. The absence of pronounced congestions of the internal organs and also of dropsy, in spite of the most intense cyanosis, may possibly be explained by structural changes with distention of the superficial capillaries and by hypertrophy of the veins with their adaptation to the slowly developing overpressure.

In congenital valvular defects, cyanosis may make its appearance in intercurrent affections which weaken the circulation, as scarlet fever, measles, whooping cough, typhoid and pulmonary affections. Other factors, as pregnancy, which increase the demand upon the heart, may produce the same effect, and lead later to cyanosis. In some cases there is transitory cyanosis in crying or coughing spells, or after bodily exertion. It may become permanent when there is inefficiency of the

cardiac muscle, or there are changes in the lungs, especially in the pulmonary vessels. Even when cyanosis has been caused by factors interfering with the circulation, there is often considerable disproportion between the cyanosis and the causative factor, as bronchitis. From a diagnostic point of view, therefore, the appearance of cyanosis in the course of slight infantile affections, as measles, and in the absence of other symptoms, especially dyspnœa, suggests the idea of congenital anomalies of the heart or large vessels.

The real cause of cyanosis in congenital cardiac defects is not yet sufficiently clear. When the two ventricles communicate with each other, there is an admixture of the venous blood entering the right ventricle with the arterial blood of the left, and the same condition results if the aorta and pulmonary artery communicate. Such a mixture may easily become venous in the peripheral capillaries, even if the motor power of the heart and the rapidity of the circulation are normal. When it is considered, however, that serious defects of the septum, even complete absence of it, does not cause cyanosis during the first years of life, the fact that the blood becomes abnormally intermixed should not be overestimated, as the cause of cyanosis. Much more important is the inefficiency of the cardiac muscles, especially of the right ventricle, which also interferes with the supply of the left ventricle and causes the circulation in the capillaries to slacken.

STENOSIS OF THE PULMONARY ARTERY AND
INSUFFICIENCY OF THE TRICUSPID

The congenital cardiac defects which are accompanied from birth by cyanosis and disturbed cardiac function are in most cases complicated by stenosis of the pulmonary artery and insufficiency of the tricuspid, so that the cyanosis is explainable by simple stasis of the venous blood.

OPEN FORAMEN OVALE

At autopsy, an open foramen ovale is frequently discovered, and yet during life there were never any indications of disturbed circulation in the cardiac cavities. To enable the blood to flow over from the right into the left auricle, the pressure in the right auricle has to be raised, as happens in tricuspid insufficiency and in stenosis of the tricuspid. In relative insufficiency, or in dilatation of the right ventricle with increased pressure in the right auricle, the blood may possibly flow over from the right into the left auricle. This may happen in cardiac insufficiency, as occurs in emphysema or in acute affections of the lungs. The early appearance of pronounced cyanosis in one of my cases of pneumonia, in which the autopsy revealed an open foramen ovale, may perhaps be explained in the same way. I also recall a case of typhoid, where a loud murmur at the base of the heart led to the diagnosis of endocarditis, which Skoda, a few years before, but unknown to me, had diagnosed as due to an open foramen ovale. The patient recovered.

The blood may flow over from the left to the right auricle when there is an associated mitral affection, and thereby lead to venous engorgement and deep cyanosis with a positive venous pulse.

OPEN DUCTUS ARTERIOSUS

The phenomenon of an open ductus arteriosus nearly always occurs in conjunction with other anomalies of the heart, as, for instance, pulmonary stenosis and stenosis of the aorta. Some show cyanosis with dyspnœa and intensified heart-beat from birth, and others present no symptoms whatever. Even combinations of an open ductus arteriosus and pulmonary stenosis may exist for years without giving rise to any special trouble, even cyanosis. In congenital, uncomplicated pulmonary stenosis, the most frequent of all congenital affections, cyanosis may either be absent or slight, and show temporary exacerbations under the influence of psychic excitement and intercurrent affections, or after severe confinements. Even where pulmonary tuberculosis develops in a case of pulmonary stenosis, both dyspnœa and cyanosis may be absent or present only to a limited extent.

STENOSIS OF THE AORTA

In congenital stenosis of the aorta cyanosis occurs from stasis in the left auricle, and the right ventricle. This defective development not infrequently gives rise to attacks of suffocation.

In stenosis of the isthmus of the aorta in children,

cyanosis and dyspnœa occur with laryngospasm and congestion, but there are also cases without these manifestations. Persons suffering from stenosis of the isthmus may attain quite an advanced age and die from the usual manifestations of cardiac insufficiency or sometimes from sudden rupture of the heart or aorta. A case observed by me showed symptoms of angina pectoris with cyanotic redness of the face during the paroxysms. In congenital stenosis of the aorta and arteries, dyspnœa and cyanosis develop in consequence of secondary changes in the cardiac muscle, frequently caused by bodily exertions, excessive indulgence in beer or wine, affections of the valves and of the pericardium, or by infectious diseases. Affections of the respiratory system, especially an acute bronchial catarrh, may cause considerable dyspnœa.

TRANSPOSITION OF THE LARGE VESSELS

Contrary to the defects of development mentioned, nearly all cases of congenital stenosis and insufficiency of the tricuspid are accompanied by cyanosis, which is explained by a simple engorgement of the venous blood in precisely the same way as is the affection of the valve acquired in later life. Cyanosis, too, is one of the most constant symptoms in the transposition of the large vessels. Other pathological manifestations like dyspnœa and attacks of suffocation, which occur so frequently in pulmonary stenosis when cyanosis increases, may be totally absent in these cases. In uncomplicated cases the cardiac sounds are pure and

the absence of cardiac hypertrophy is diagnostically important. Cyanosis in the transposition of arteries may be explained not only by engorgement, but also by the venous condition of the blood circulating in the systemic arteries. The tendency to spasms associated with errors of development is attributed to defective supply of the brain with arterial blood.

MITRAL STENOSIS (CONGENITAL)

A special group in the symptomatology is composed of those forms of congenital, strictly mitral stenosis, to which Duroziez first called detailed attention. These are the special forms of mitral stenosis in which the orifice is an elastic funnel, and the edge of the valve is smooth and without vegetations. A characteristic clinical picture corresponds with these autopsy findings of a purely mitral stenosis without vegetations on the valve. It is a striking fact that no etiology can be adduced for these cardiac defects, as the anamnesis reveals no articular rheumatism, scarlet fever, chorea or angina. It appears, however, that these patients in their very early infancy were pale and weak, and that the slightest efforts induced paroxysms of palpitation and dyspnœa. One or the other predominated, or, what is more generally the case, both manifestations occurred with equal intensity. These forms of mitral stenosis, have in some, a small left ventricle and in others both ventricles are hypertrophied. They must be regarded as congenital defects of development of the mitral orifice with hypoplasia of the arteries, as the

functional symptoms. Especially dyspnœa and palpitation often date from earliest infancy. They appear at times only in advanced life, but in girls usually between the fifteenth and twentieth year. These young women present all the symptoms of chlorosis and complain particularly about palpitation and dyspnœa. Auscultation reveals the sign of a pure mitral stenosis, presystolic murmur, loud first sound, accentuation and frequently reduplication of the pulmonic sound. This type is described as chlorotic mitral stenosis. At autopsy there is found pure stenosis of the mitral with no roughness of the valve, and associated with a thin, narrow, elastic aorta, with irregular arteries, and hypoplasia of the internal genitalia. While most of these cases show the ordinary symptom-complex of chlorosis, in others the cardiac conditions displays the more prominent signs: dyspnœa, stases, cardiac arrhythmia and edema. These affections, especially during pregnancy, may lead to grave difficulties in breathing, and not infrequently to rapid death from pulmonary edema. Some cases present a picture which, on account of the emaciation, pallor, cough and tendency to hemoptysis, resembles more pulmonary tuberculosis than a cardiac defect. A pregnant woman brought into the clinic in a moribund condition exhibited numerous râles audible over both lungs, and gave the general impression of phthisis. The pulmonary edema subsided and auscultation disclosed the presence of a pure mitral stenosis. Etiology of any kind was not obtainable, but the anamnesis showed that the pa-

tient as a child was always pale and dyspnœic, and at the age of puberty was chlorotic with anomalies of menstruation. The anamnesis, together with the objective findings, justified the diagnosis of a congenital mitral stenosis.

From these explanations about the congenital cardiac defects, it follows that there are numerous cases of congenital cyanosis in which dyspnœa and cyanosis do not follow a parallel course, and that the former may sometimes be totally absent. Possibly this tolerance on the part of the center for respiration points to a reduced requirement for oxygen and corresponds with the reduced body temperature, apathy, drowsiness and the other signs of bodily and mental inhibition of development so often observable in these patients. The respiratory center, having lost its excitability, does not seem to react well to the poorly oxygenated blood and to the dyspnœic irritations. In the vast majority of cases, however, there is side by side with pronounced cyanosis also dyspnœa, which, from slight causes, may become intensified to the point of suffocation, and is often associated with palpitation of the heart, oppression, fainting and other signs of cardiac weakness.

II

DYSPNŒA AND CYANOSIS IN ACQUIRED CARDIAC LESIONS

MITRAL INSUFFICIENCY AND STENOSIS

In acquired mitral insufficiency of varied but mainly rheumatic origin, also in mitral stenosis, dyspnœa is a frequent occurrence, especially in the stage of broken compensation on account of the congested lungs. It is characterized by râles with their greatest intensity at the base. The dyspnœa may attain the intensity of orthopnœa. An upright position is advised to diminish the congestion of the brain and to aid the circulation of the oblongata. Insomnia in these patients is also explained by the mechanical congestion of the brain.

AORTIC LESIONS

In contradistinction to affections of the mitral valve in which cyanosis is an early or late symptom due to venous engorgement, diseases of the aortic valve are usually distinguished by the absence of cyanosis, so that there is a certain justification for speaking of a blue facies mitralis and a pale facies aortica. The mechanism of the dyspnœa in aortic conditions is also different. In affections of the mitral valve, there is, beside the congestion of the lungs, also a stasis in the oblongata which increases upon lying down, owing to the retarded flow of blood, and compels patients to

select a more upright posture in bed. In insufficiency of the aortic valve, however, the important dyspnœic factor is the ischemia of the oblongata. There is, as it were, the *pulsus celer* which poorly nourishes the center of respiration. In some cases the periaortic processes and the irritation they exert upon the branches of the vagus have a certain importance in the causation of the asthmatic attacks. This also explains why in the dyspnœa and cyanosis of mitral affections, digitalis is an especially successful remedy, while in the dyspnœa of aortic insufficiency sedative remedies like potassium bromide and opium preparations are more effective. Cyanosis, however, occurs also in insufficiency of the aortic valve, if there are complicating affections of the mitral or if bulging of the interventricular septum due to marked excentric hypertrophy of the left ventricle restricts the space of the right ventricle, which in its turn becomes hypertrophied. If fatty degeneration of the myocardium makes the left ventricle inefficient, or if complicating affections of the respiratory system and of the serous membranes as pericarditis develop. In aortic stenosis, therefore, unless it attains a high degree and becomes complicated, neither dyspnœa nor cyanosis is present in the pathological picture.

AFFECTIONS OF THE PULMONARY VALVE

Cyanosis is not a constant symptom in affections of the semilunar valve of the pulmonary artery. In acquired insufficiency of the pulmonary valve without

stenosis, which rarely occurs from rheumatic endocarditis, and somewhat more often from trauma, like a blow on the chest, cyanosis appears in the beginning of the disease, less prominently than dyspnœa, even if the patient remains absolutely quiet. In the further course, cyanosis follows insufficiency of the right ventricle, as in other cardiac defects, or more frequently embolism of the pulmonary artery. In acquired stenosis of the pulmonary artery cyanosis may be completely absent or makes its appearance at an advanced stage of the affection. The dyspnœa is slight even after bodily exertion as compared with the frequency of hemoptysis. The explanation is that the manifestations are caused less by the valvular defects than by the frequent combination with tuberculosis.

ACUTE ULCERATIVE ENDOCARDITIS

In acute ulcerative endocarditis, dyspnœa and cyanosis depend less on the primary affection than on its many complications, as valvular aneurisms, cardiac aneurisms, thrombosis. They are also affected by the localization of the lesion at the various valves and their orifices, on the degree of cardiac inefficiency and the rapidity of its development, on the simultaneous involvement of the myocardium and pericardium, and finally on the site of the metastases especially of multiple emboli of the lung in affections of the right heart. The bacterial endocarditis which occurs in the course of chronic pulmonary tuberculosis has a special tendency to dyspnœa and hemoptysis. As a rule dyspnœa

is but slightly pronounced in pure endocarditis, and is in many cases experienced only as an oppression of the chest, which may increase through psychic excitement and by sitting up in bed. The occurrence of intense dyspnœa therefore requires careful examination, especially of the organs of respiration. Cyanosis, like dyspnœa, plays a small part in the clinical picture of endocarditis. Septic forms of malignant endocarditis may show pronounced anemia from the onset, and may be mistaken for pernicious anemia (*febris pallida*.)

PURULENT AORTITIS

In the acute forms of purulent aortitis, where the abscess perforates the intima of the aorta and empties into the circulation, retrosternal pains, girdle sensation around the thorax, pulsation of the distended aorta and especially paroxysmal dyspnœa with prolonged inspiration, and relatively slight difficulty in expiration, may be observed. Intense dyspnœa and cyanosis with fear of death may occur in endocarditis affecting the aortic valves if the vegetations on the valves occlude the aortic orifice. In two cases of pneumonia under my observation, in which insufficiency of the aortic valve developed, the pulse became slower, smaller and harder, and a systolic murmur, heard over the aorta, in addition to the diastolic one, grew continually more protracted and distinct, a sign of progressively developing stenosis. The rapidity of the pulse disappeared, it became continually smaller, and suddenly, with violent manifestations of asphyxia and general epileptiform

convulsions, the patients died from occlusion of the aortic orifice.

PERICARDITIS AND ADHESIVE PERICARDITIS

In pericarditis, dyspnœa and cyanosis are inconstant phenomena. Dyspnœa may be absent, slight, or increased to a most intense degree, orthopnœa, with the sensation of suffocation. This depends, above all, upon the quantity of the exudate, the rapidity of its formation, the condition of the myocardium as well as upon complications of the lung and pleura, and partly, also, on the primary affection, which caused the pericarditis. In large exudates it is caused by mechanical obstruction to the flow of blood into the auricles and consequent defective filling of the ventricles. To this is added the direct compression of the vascular trunks especially of the superior vena cava, the more so if the pericardium is distended by a rapidly developing effusion. Just as in extensive pleural exudates, dyspnœa may be slight as long as the patient remains quiet in one position, so at times marked exudates into the pericardium do not cause any material difficulty in breathing. There is often a disproportion between the intensity of the dyspnœa and the quantity of exudate in tuberculous pericarditis. As the tuberculosis spreads from the neighboring organs—pleura, lungs, peritoneal and mediastinal glands—the dyspnœa may also emanate from an affection of these structures. Pericarditis in the course of miliary tuberculosis or tuberculosis of the serous membranes sometimes does not show any material increase in dyspnœa. In some cases of

isolated tuberculosis of the pericardium, dyspnœa may, however, attain an excessive degree. This holds good for those forms of pericarditis which sometimes occur after slight pneumonia, and are frequently overlooked. The appearance of dyspnœa with cyanosis in these cases should direct attention at once to the pericardium.

Dyspnœa in pericarditis may, therefore, be due to various causes. In some cases, the pains from involvement of the neighboring pleura may cause dyspnœa; perhaps the irritation of the vagus plays a rôle, in others it may be an affection of the phrenic nerve. Where there is considerable exudate, the pressure on the auricle and pulmonary veins, as well as on the left lung, may cause immobilization of the diaphragm by paralysis or downward displacement. Very large exudates may restrict the thoracic space for all structures. Increase of dyspnœa in the dorsal position and on the left side compels patients instantly to assume an upright posture or to bend forward, and to the left, in order to relieve the pressure on the organs lying posterior, especially the vena cava superior and the right auricle, and possible also the left bronchus. This certainly presupposes a certain mobility of the exudate, which, in pericardial exudates with partial adhesions, is not always present.

Adhesions of the heart to the pericardium, which frequently exist without causing any symptoms, are accompanied by dyspnœa when involvement of the muscular substance follows inefficiency of the left ventricle, and by cyanosis, with manifestations of conges-

tion and dropsy often of extreme degree, if the right ventricle is prominently involved, as in tricuspid insufficiency.

In consequence of the compression of the auricles and the venæ cavæ, and also from cardiac inefficiency through involvement of the myocardium in the inflammatory process, we find associated with the greatest accumulation of fluid in the pericardium, deep cyanosis, edema of the liver and lower extremities, pulse hardly perceptible and paroxysms of extreme dyspnœa. In the subacute or chronic pericardial exudates of anemic (tuberculous) subjects, the exudate is generally hemorrhagic, and the cyanosis in spite of considerable dyspnœa and orthopnœa stands either quite in the background or is only indicated by a livid coloration of the lips and mucous membranes. In these pericardial effusions, however, which are a part manifestation of miliary tuberculosis, cyanosis usually shows a considerable increase in intensity. In the acute exanthemata, especially variola and scarlet fever, cyanosis with a greatly accelerated pulse should direct attention to an existent pericarditis. The same holds good for pericarditis occurring in the course of pneumonia, especially in alcoholics; also for those forms of pericarditis which develop in the course of chronic affections, as Bright's disease, scorbutus and carcinoma.

HYDRO-, HEMO-, AND PNEUMOPERICARDIUM

In hydropericardium, dyspnœa and cyanosis partly depend upon the primary affection, like Bright's dis-

ease and cardiac defects. Large accumulations of fluid are able to produce cyanosis and dyspnœa by compression of the lungs and auricles as in pericarditis, or to augment what already exists. Dropsical transudates into the pericardium, in contrast to pericardial exudates, are always changing and the congestion is correspondingly dependent, increasing or decreasing with each change of position, upon the stronger or weaker compression of the auricles, and these changes will influence the appearance and intensity of the dyspnœa and cyanosis. These symptoms varying in the upright and dorsal positions, may direct attention to the existence of an hydropericardium and demand the examination of the cardiac dullness and cardiac impulse with the body bent forward. This may also be important in simultaneous emphysema, the presence of which makes the diagnosis of hydropericardium difficult.

Effusions of blood into the pericardial sac (hemo-pericardium) due to rupture of aneurisms of the aorta, heart or coronary arteries, tearing of the myocardium at fatty and necrosed places, trauma from without or swallowing pointed objects, also in rare cases to perforation of tuberculous glands, lead to death with dyspnœa, pains in the cardiac region, fainting, anemia and convulsions. If the course is protracted owing to the small size of the tear through which the blood oozes out slowly into the pericardium, cyanosis or livid coloration, generally with increasing enlargement of the cardiac dullness, disappearance of

the cardiac impulse and of cardiac sounds in consequence of the accumulation of blood in the pericardial sac, are of importance for the diagnosis.

Pneumopericardium occurs through injuries with pointed objects, either from without by stabbing or from within by swallowing pointed foreign bodies, by perforation of tuberculous cavities or in perforating pyo-pneumothorax; less often in ulcerating carcinoma of the esophagus and in ulceration of the stomach into the pericardium. In this condition dyspnœa, sensation of oppression in the cardiac region, also cyanosis in consequence of the compression of the heart and lungs, are constant manifestations. The characteristic phenomena of auscultation and percussion and their variation, as the patient changes his position, make the diagnosis safe.

FATTY AND MUSCULAR DEGENERATIONS

Dyspnœa is a frequent symptom of fatty heart, as in valvular lesions, indicating defective motor power of the heart and inefficiency of the left ventricle. This dyspnœa may at times increase after bodily exertion. The pulse frequency is considerably accelerated and there may be orthopnœa with cyanosis and a sensation of fear and oppression.

Also other affections of the muscular substance of the heart, partly primary and in part secondary to interstitial or parenchymatous inflammations after infectious diseases, weaken the cardiac power and thus occasion dyspnœa and cyanosis. There are a few

infectious diseases especially which are associated with the signs of an acute or chronic affection of the heart, both during their febrile course and in convalescence. In this manner dyspnœa and cyanosis develop in the course of an acute articular rheumatism, not only in the hyperpyretic form, but also in cases with low temperature, in whom, however, cerebral symptoms and delirium occur during the first few days. Salicylic preparations are administered without effect, cardiac dullness increases and the pulse becomes small and irregular. Autopsy in these cases reveals the valves intact, the cardiac cavities much distended and the muscle excessively friable. Also in diphtheria there are similar degenerations of the cardiac muscle and of the coronary vessels, both during and after the acute stage, and they are not infrequently the cause of sudden death. The same is true in scarlet fever, dysentery, and typhus fever. So far as the latter is concerned, dyspnœa and cyanosis occur even during convalescence from insignificant causes, and this is a sign of the involvement of the heart and arteries. The changes of the myocardium and of the vessels in enteric fever are recognized both during the course of the disease and in convalescence by various clinical signs of cardiac weakness, as cyanosis, puffy face, edema of the ankles, weakness of the first mitral sound, arrhythmia with low blood pressure, slight diuresis and not infrequently albuminuria. These manifestations may sometimes lead to early and even sudden death, but on the other hand they may pass off

quickly and without any evil consequences. The occurrence of the embryonic heart rhythm with cyanosis of the face and extremities, diminution of diuresis, lowering of the skin temperature with elevation of the body temperature are, as a rule, preagonal symptoms.

Cyanosis is also observed in rare affections of the heart: amyloid degeneration, cardiac abscess, also in cardiac tumors: carcinoma, sarcoma, tubercles, syphiloma, actinomycosis; and in parasitic affections like cysticerci and echinococci.

THROMBOSIS OF THE HEART

Intense dyspnœa, frequently accompanied by bloody expectoration, and rapidly increasing cyanosis with coldness of the extremities is observed in thrombosis of the heart. Thrombi situated in the left auricle and causing stenosis of the mitral orifice, or right-sided cardiac thrombi migrating into the pulmonary artery, will, as a matter of course, be associated with dyspnœa and cyanosis. In extensive thrombi of the left auricle pressure from without may diminish the lumen of the pulmonary artery, in thrombi of the right auricle that of the aorta, and both give rise to systolic stenotic murmurs and deficient filling of the corresponding vascular systems. While many cases exhibit considerable dyspnœa and cyanosis, there are on the contrary cases of cardiac thrombosis without cyanosis and without material dyspnœa, in which manifestations of defective filling of the arterial system are evident; as a very frequent, small, irregular, radial pulse, great pallor, sensation of fear and fainting attacks. In other

cases, symptoms arising from distant organs dominate the clinical picture. In a patient under my observation with carcinoma of the stomach, thrombosis of the left heart occurred at an atheromatous mitral valve and gave on auscultation the signs of mitral insufficiency, which led to a fatal embolus in the artery of the fossa of Sylvius. Manifestations on the part of the stomach and intestine, as emboli of the intestinal arteries, appear in other cases. Nevertheless, in the majority of cases cardiac manifestations are pronounced. In our clinic a waiter, twenty-seven years of age, suffered from dilatation of the heart, in the stage of ruptured compensation, ascites and anasarca. Patient died within a few days, after increasing dyspnœa and cyanosis, palpitation, gallop rhythm, reduction of blood pressure and reduplication of the second pulmonic sound. The autopsy showed atheroma of the coronary vessels with the formation of extensive cardiac indurations in the area of the left ventricle (*aneurisma partiale cordis*) and correspondingly distended parietal thrombosis.

THEORIES OF CARDIAC DYSPNŒA (BASCH'S AND FRAENKEL'S)

According to Basch, cardiac dyspnœa occurs in the first place from enlargement in the volume of the lung, associated with congestion which he designates as pulmonary rigidity (*Lungenstarrheit*). Owing to the inefficiency of the left ventricle and the correspondingly greater activity thrown upon the right ventricle, the lung becomes rigid, distends only with difficulty,

and thus forms an impediment to respiration. Cardiac dyspnœa owes its origin to this mechanical obstruction, not to a neurotic origin occasioned by the chemical properties of the blood.

A. Fraenkel attaches great importance to the engorgement of the bronchial mucous membrane in the development of cardiac dyspnœa and considers the participation of a secondary bronchospasm not entirely excluded as a factor in the paroxysms of cardiac asthma. Fraenkel's theory explains more simply the suddenly appearing resistance to the circulation than does Basch's pulmonary rigidity, although the latter's experimental investigations and explanatory statements concerning the conditions of blood pressure and circulation in cardiac asthma are of fundamental importance for the understanding of many phenomena occurring in connection therewith.

Basch's doctrine of secondary inefficiency of the left ventricle as a sequel to increased intracardiac pressure which originates under the influence of high arterial pressure, explains why the gravest paroxysms of cardiac dyspnœa may occur not only with low blood pressure as in mitral stenosis, but also with normal or even increased arterial pressure. The left ventricle, as it were, becomes tired in the struggle with the increased powers of resistance on the part of the arteries, and the consequence of such secondary inefficiency of the heart is increased pressure in the left auricle and the pulmonary capillaries.

The inefficiency of the left ventricle occurs more

readily if the heart is already in a badly nourished state and if the demand made upon its power is great. This kind of dyspnœa, occurring as it does if the left ventricle has to overcome considerable obstruction, happens very frequently in arteriosclerosis and in contraction of the kidneys.

FINAL CAUSE OF CARDIAC DYSPNŒA

Since the final cause of dyspnœa in affections of the heart is inefficiency of the left ventricle, and is due to the fact that the latter does not completely empty its contents into the arteries and thus causes engorgement of blood in the left auricle and lungs, the pulse in the fully developed paroxysm of cardiac asthma, although perhaps strong enough at first, becomes softer and smaller. There may, however, be elevation of the blood pressure under the influence of the blood in dyspnœa, exciting the vasomotor centers. The increased tension of the pulse may even continue during the paroxysm together with dyspnœa and especially cyanosis, particularly if the primary affection is associated with abnormally high blood pressure.

DYSPNŒA AND BLOOD PRESSURE

There are cases in which the dyspnœa improves under falling blood pressure, and also others in which the dyspnœa decreases although the blood pressure rises. The latter is, for instance, the case after the administration of digitalis in mitral stenosis. Digitalis, as a rule, has the effect of increasing the arterial

pressure. The causes vary: increase of cardiac power, lengthening of the systole and constriction of the vessels. Occasionally, however, the cardiac function improves under digitalis in both systole and diastole, while the pathologically increased blood pressure diminishes. In our clinic there have been several cases with high blood pressure, dyspnoea and cyanosis, in which the blood pressure after the administration of digitalis fell, the dyspnoea and cyanosis subsided, while the other phenomena of stasis, such as albuminuria, remained practically stationary. Here it must be assumed that in consequence of the better contraction of the left ventricle and the strengthening of the right ventricle of the heart, the carbon dioxid tension was decreased by the improved arterialization of the pulmonary and coronary blood, thereby lowering the excitation of the center of respiration. This is the condition in arteriosclerosis, where there is cyanosis with high blood pressure: the state which Sahli describes as high-pressure congestion. Traube pointed out that digitalis, administered in such cases in order to mitigate the symptoms, may expose the patient to the danger of cerebral hemorrhage. These apprehensions are certainly justified when it is considered that generally digitalis increases the arterial pressure and that a possible increase of pressure throws an additional task upon an already inefficient heart. Sahli, on the other hand, made the important statement at the Nineteenth Congress for Internal Medicine in Berlin, 1901, that digitalis was capable not only of re-

moving the engorgement in high pressure congestion, but also of considerably reducing the high blood pressure, instead of further increasing it. This paradoxical result of digitalis was partly explained by Sahli's statement that there was a lessening of the vascular spasm by removal of the dyspnœa, and partly by the assumption that the enlarged digitalis pulse was capable of overcoming the peripheral obstruction in the finest arteries, thus dilating the vessels. Based upon these experiences, Sahli has pronounced the dictum that high arterial pressure in itself is no contra-indication to the administration of digitalis. Our own experience entirely agrees with that of Sahli in this respect, for we, too, have had cases of arteriosclerosis with dyspnœa, considerable cyanosis and increased blood pressure, in which the latter sank, dyspnœa decreased and cyanosis disappeared after the administration of digitalis.

III

DYSPNŒA AND CYANOSIS IN VASCULAR LESIONS

ARTERIOSCLEROSIS AND ANEMIA

Those cases of arteriosclerosis act very differently where, although dyspnœa is associated with high blood pressure, there is anemia instead of cyanosis. If these cases are complicated with aortic insufficiency, there occurs consequent upon the dilatation of the abdominal vessels an aspiration of the blood, as it were, into the latter. These patients bleed into themselves. There is an actual abdominal plethora in consequence of intravascular bleeding, while the skin and brain may perhaps be anemic in consequence of secondary vasoconstriction. The patients are as pale as if in a faint and yet they have not lost a drop of blood, and every drop is normal.

ARTERIOSCLEROSIS AND PSEUDO-ANEMIA

These forms of chronic pseudo-anemia in arteriosclerosis arise from unequal distribution of blood, shown by hyperemia of the abdominal vessels and complemental anemia of the skin and are possibly caused by dilatation of the intestinal vessels, consequent upon irritation of the depressor fibers of the

vagus. Paralysis of the vasomotors of the splanchnic nerve may also have the same effect. To this group very likely belong those cases which are observed before the deposit of inflammatory exudates in the peritoneum, as in polyserositis, perihepatitis and peritonitis associated with affections of the heart. Nevertheless, in these pale arteriosclerotic patients there may be high blood pressure in consequence of partial vasoconstriction. If in these cases digitalis is administered on account of dyspnœa, our experience shows that, in contrast to the cyanotic forms, the blood pressure rises and the dyspnœa increases. The same result is experienced in aortic insufficiency, with excessive pallor of the skin and signs of engorgement of the abdominal organs. These pale cases of aortic insufficiency are therefore not suitable for digitalis treatment. Their dyspnœa would be intensified with increasing blood pressure, while by way of contrast, the blue cases of aortic insufficiency, in which dyspnœa is associated with cyanosis, occasionally react to digitalis with beneficial results.

ARTERIOSCLEROSIS AND THE HEART

In the vast majority of cases dyspnœa in arteriosclerosis is caused by temporary or permanent inefficiency of the left ventricle, while the dyspnœic paroxysms are associated with stasis in the lungs, pulmonary râles and sometimes with albuminous expectoration tinged with blood as in cardiac asthma. On the other hand, there are cases in which there is a

surprising disproportion between the intensity of the dyspnœa and the physical manifestations in the lungs. "Dry" dyspnœa, without expectoration, in which the vesicular respiration is diminished, with only very few if any dry râles, recalls in a way bronchial asthma, from which, however, it is chiefly distinguished in that inspiration and expiration are almost uniformly prolonged. In most cases, however, there is a combination of both affections, in the sense of a composite of cardiac dyspnœa and bronchial spasm. Possibly some periaortic processes, involving branches of the pneumogastric nerve may here play a rôle. This must at least be assumed for those cases of asthma, accompanied by stenocardiac paroxysms and not based on coronary sclerosis, that they be interpreted as angina plexus.

This enumeration by no means exhausts the causes of dyspnœa in arteriosclerosis. Its appearance is in the first place dependent upon the question whether the heart, which compensates for the diminished performance of the arteries, is hypertrophied, and how long it will remain capable of functioning in this condition. The sclerosis of the arteries may, as is well known, be irregularly disseminated. It has often been observed that considerable change in the arteries of one organ are not always accompanied by equally important change in those of another. Therefore, the occurrence and the polymorphism in the varieties of dyspnœa depend upon the localization in those organs whose disturbances of circulation and nutrition react upon the

condition of respiration and circulation. In regard to localization in the respiratory apparatus, the intercostal and bronchial nutrient arteries should be considered, and in regard to arteriosclerosis of the heart, both coronary arteries. When it is remembered that numerous branches arise from the descending aorta; the posterior bronchial, the esophageal, the mediastinal and especially the intercostal arteries, which supply both the ribs and costal muscles, it is clear that an extensive sclerosis of the thoracic aorta with constriction at the points of radiation of these small branches, will lead to a defective blood supply of the corresponding regions. Atheroma of the aorta, therefore, may lead to disturbed nutrition of the bronchi, lungs, ribs and muscles. In this manner secondary emphysema with atrophy of the lungs and thorax, may result, and both this condition as well as secondary bronchial catarrh may have to be taken into account as etiological factors for dyspnœa.

The significance of arteriosclerosis for the heart is evident since the functional activity of the latter depends upon whether the coronary arteries, which nourish the heart, remain intact or are involved at an early period. In the latter case, the hypertrophy of the heart is checked in spite of increased peripheral obstruction, caused by the sclerosis of the trunk arteries, and there results a general degeneration of the myocardium or partial myomalacia with its sequelæ, as aneurism of the heart and the formation of indurations. In these cases a clinically well characterized

symptom-complex may dominate the pathological picture, *i. e.*, angina pectoris coronaria. In pure cases of coronary angina both dyspnœa and cyanosis are absent. In spite of the oppression of the chest, respiration is either not changed at all or only slightly deeper or more frequent. Sometimes there is even apnœa, when the patient immobilizes the thorax in consequence of great pain. The face is as a rule pale during the stenocardial attacks. In other cases, there is cyanosis of the lips which stands out in striking contrast to the pallor of the skin. There are also combinations of angina pectoris with cardiac asthma, in which stenocardial symptoms occur with dyspnœa. If the course of the affection becomes protracted, there will also be intense cyanosis with acute emphysema, congestion of the lungs and bloody expectoration. The acute exacerbations of chronic aortitis and especially of periaortitis may, likewise, exercise an influence upon the cardiac plexus and its pulmonary fibers and lead to death from asphyxiation through acute or subacute edema of the lungs, under manifestations of severe dyspnœa and cyanosis with aggravated and protracted inspiration.

Dyspnœa resulting from localization of arteriosclerosis in the circulatory apparatus, especially in the heart, depends, as may easily be understood, on inefficiency of the left ventricle, with increased pressure in the left auricle, and later stasis of the lungs. In these cases the blood pressure is low, unless dyspnœa and defective arterialization of the blood cause a vascular spasm by increased vasomotor irritability and with it

an augmentation of the resistance. This form of dyspnœa is accompanied by cyanosis, and, as explained, the increased blood pressure is a prognostically favorable sign if the dyspnœa decreases under the influence of therapeutic measures. In cases where inefficiency of the left heart is developed under the influence of high aortic pressure, there will be dyspnœa in consequence of the increased pressure in the pulmonary vessels, if the heart is unable to overcome the resistance. The right heart is compelled to empty its contents into the pulmonary artery with great force. If the right ventricle also becomes inefficient, and dilates, there may be distention of the valvular ring, and the tricuspid valve becomes incompetent. The insufficiency of this valve is equivalent, to a certain extent, to venesection of the pulmonary artery or rather of the pulmonary circulation. This causes the blood pressure in the pulmonary area to sink, the formerly accentuated second pulmonic sound becomes weaker, and the cardiac dyspnœa frequently diminishes, while cyanosis with manifestations of congestion increases in the area of the vena cava. This decrease of dyspnœa by the opening of the safety valve, namely, the tricuspid orifice, with simultaneously increasing cyanosis, does not last long, because the cyanosis of the brain, medulla oblongata and kidneys, as well as the engorgement of the blood in the coronary veins, produce new factors for causing dyspnœa which is then not of cardiac, but rather of cardio-bulbar or cardio-renal origin.

ARTERIO-SCLEROSIS AND THE KIDNEYS

The determination of the localization of the condition in the kidneys is of great importance among the myriad causes of dyspnœa in arteriosclerosis. As the arteriosclerotic affection develops in the kidneys, the latter become inefficient. The consequence of this inefficiency is a retention of toxic metabolic products which are able to exert an irritation upon the oblongata, the pulmonary vessels, and perhaps also on the bronchial muscles. The accumulation of oxydizable metabolic products should not be underrated, as they prey upon the oxygen and lead to improverishment of the blood and tissues in oxygen. These factors may produce dyspnœa (*sine materia*) in the sense of Charcot, in which dyspnœa and oppression bear no relation to the physical signs of the heart and lungs or to the findings at autopsy. This form of dyspnœa is to be traced back to direct irritation of the respiratory center, and is therefore purely toxic and of bulbar origin, resembling uremia by its accompanying manifestations, as fibrillary twitchings, exaggeration of the tendon reflexes, formication of the fingers and toes, vertigo, tinnitus aurium, headache, polyuria, etc. The occurrence of albuminuria, of renal elements in the urine and the reaction of the renal affections upon the heart, depend above all upon the degree of injury to the renal tissue. It should not be overlooked, however, that in old people the arteriosclerotic affection of the kidneys, where they are no pronounced symptoms of nephritic disorder, may give rise to dyspnœa which

is sometimes expressed only by polypnœa with deepening and acceleration of the movements of respiration. Huchard describes a special form of toxic alimentary dyspnœa which occurs in the beginning of arteriosclerosis. This stage is called the presclerotic and is characterized by permanent elevation of the blood pressure. In these forms of dyspnœa there are functional disturbances of the renal secretion, occasioned by spasm of the renal vessels. There is intermittent dysphagia, an intermittent claudication, such as also occurs in other organs in consequence of deficient circulation and constriction of the vascular lumen. The renal inefficiency is principally exhibited by defective excretion of toxins which are elaborated from the food and which produce paroxysms of dyspnœa, especially at night. The cause of their occurrence at night is explained according to French clinicians by the accumulation of toxins in the blood at night. This is based on the investigations of Bouchard, who determined that the toxicity of urine excreted at night is diminished. Importance is also attributed to the effect of the toxins upon the vessels, which leads to increase of the blood pressure. The final result of autointoxication, therefore, is a vascular spasm and the mechanism of dyspnœa is the same as is observed in the increase of blood pressure from other causes due to increased demands upon and secondary inefficiency of the left ventricle. Toxic alimentary dyspnœa deserves special interest, because it can be successfully treated by withholding meat and replacing it by a lacto-vegetable diet.

CEREBRAL AND SPINAL ARTERIOSCLEROSIS

In contrast to the bulbar form of dyspnœa of toxic origin, those forms should be mentioned which result from the localization of the arteriosclerosis in the brain and medulla oblongata. These are disturbances of respiration which accompany Stokes-Adams disease and in which ischemia of the oblongata is the most important factor in the pathological picture. The Cheyne-Stokes' respiratory phenomenon, which is so frequently observed in the cerebral and renal localization of arteriosclerosis, also belongs to this group. Other forms of paroxysmal dyspnœa which occur under the picture of sudden, sometimes periodically intermittent, asthmatic attacks, with normal function of the heart and kidneys in pale arteriosclerotic subjects, and which defy all therapy with the exception of morphin, should also be included. Huchard has observed such cases and considers them due to sclerosis of the bulbar arteries.

SIGNIFICANCE OF DYSPNŒA IN ARTERIOSCLEROSIS

It will thus be seen that dyspnœa in arteriosclerosis may be produced by various factors; central local irritation of the oblongata, toxic and uremic influences in renal insufficiency, irritation of the pneumogastric nerve in peri-aortic affections, secondary emphysema and bronchitis. The most frequent cause of dyspnœa is inefficiency of the heart, the contributing factor to which may, in a large number of cases, be found in an increased blood pressure. In all forms of dyspnœa

with high blood pressure, the object of the therapy is to reduce it, and in these cases decrease in the dyspnœa corresponds to the fall in the blood pressure. All factors which raise the blood pressure may disturb the cardiac equilibrium and produce acute paroxysms of cardiac asthma. In this connection a case mentioned by Basch is interesting, because, after the administration of atropin to correct arrhythmia, an asthmatic paroxysm occurred with tachycardia, palpitation and increase of the blood pressure.

Not every arteriosclerotic patient with high blood pressure is dyspnœic. There are individuals with very high blood pressure who do not for years exhibit any dyspnœa or cardiac weakness, if the heart remains efficient and accustoms itself to the increased task. The fall of the blood pressure during the dyspnœa of arteriosclerosis may have different meanings, favorable and unfavorable. The fall is unfavorable, if, owing to diminished cardiac power, the left ventricle expels too little blood into the aorta. The pressure in the left auricle and pulmonary vessels rises, while dyspnœa and cyanosis increase. But there are cases where the blood pressure sinks and respiration becomes free, but the general condition nevertheless is worse. That happens when complications occur, such as hemorrhages or secondary anemia with quantitative diminution of the blood, splanchnic engorgement to which reference has already been made, ascites and dropsy, in subjects with sclerosis of the visceral region, due to a diminished *vis a tergo*. So far as prognosis is concerned, therefore,

neither the abatement of the dyspnœa nor the reduction of the vascular tension can always be interpreted as an improvement of the cardiac function.

From these observations on dyspnœa in cardiac defects and arteriosclerosis, it follows that dyspnœa may be produced from various causes and causative factors, whether it is dyspnœa from exertion (*Dypsnoé d'effort*), or paroxysmal dyspnœa, associated with oppression and fear, the true cardiac asthma. In order to form a judgment on the nature and causes of dyspnœic paroxysms in a given case, the anamnesis and accompanying symptoms should be taken into consideration in addition to a careful examination of the patient. In one case the manifestations of the brain or oblongata are decisive, as we have seen in Stokes-Adams disease and the Cheyne-Stokes' phenomenon; in another case it is that form of dyspnœa which may be followed by a graphic curve of respiration; in others again the pulmonary findings by auscultation may assist, as for instance, the signs of catarrh decreasing from the base of the lungs to the apices, as observed in the dyspnœa of stasis. In many cases examination of the sputum may reveal heart failure cells, eosinophile cells, erythrocytes and Curschmann's spiral; in still others the examination of the heart itself may demonstrate enlargement of cardiac dullness, weakness of the heart sounds, or the reverse. The relation to blood pressure has already been considered. The various forms of arrhythmia and of gallop rhythm which accompany dyspnœa should be subjected to a special

analysis. In one case the pulse during a paroxysm of dyspnœa is irregular, soft and compressible; in another retarded, full and hard.

ANEURISM OF THE AORTA

Dyspnœa and cyanosis in aneurisms of the aorta are symptoms of compression and therefore dependent upon the size and site of the aneurism. Compression of the right auricle and of the large venous trunks cause the most intense cyanosis, which may attain an exceedingly high degree in thrombosis, endophlebitis and especially in perforation of the aneurism into the veins. Cyanosis together with dyspnœa occurs when the pulmonary artery is exposed to pressure from aneurisms and there is consequent hypertrophy of the right ventricle. The compression of the aorta results in a similar way, and in large aneurisms also the uncommon compression of the pulmonary veins. The most frequent cause of dyspnœa is compression of the trachea and bronchi, but there may be contributing factors in the constriction of the thoracic space from compression of the auricles and the pulmonary vessels, also from the work thrown upon the diaphragm by large aneurisms. Finally various complications, such as inflammation of the pleura, pericardium and mediastinum play a part in the etiology of dyspnœa. Pains, too, occasioned by compression of the intercostal nerves of the brachial plexus, may act in a similar way. Dyspnœa and cyanosis in the final phase of aneurisms, as in other cardiac affections, are per-

fectly explained by the inefficiency of the cardiac function. Compression of the pneumogastric, phrenic, and recurrent laryngeal nerves may produce symptoms which recall asthma nervosum, the asthmatic paroxysms of emphysema and hysterical asthma. In these cases it should be remembered that the asthmatic paroxysms in aortic aneurisms belong, as a rule, to the type of cardiac asthma and that dyspnœa occurs in both phases of respiration; the sputum is rich in cells, often even pigment cells. The dyspnœa outlasts the attack and recurs after bodily exertion.

Dyspnœa in aneurisms may also be intermittent. Grossman is of opinion that dyspnœa, accompanied by paralysis of the vocal cords and disappearing in a relatively short time, is characteristic of aneurisms which develop in the neighborhood of the recurrent laryngeal. This condition is in contrast to the malignant endothoracic tumors where these disturbances are irreparable.

Dyspnœa with the most intense cyanosis of the face and neck with positive venous pulse and hum over the aorta, with edema of the upper half of the body, while the lower half is unaffected, occurs in perforations of aneurisms of the aorta into the superior vena cava. In perforating aneurisms of the aorta, cyanosis may be relatively absent in spite of the most marked dyspnœa and of violent pains in the cardiac region, if the bleeding takes place slowly and progressive anemia develops. This has been observed in dissecting aneurisms where the blood is retained by the vascular membranes.

Even in perforations into the pulmonary artery cyanosis may be absent. This was shown by Mager, in whose case, after the establishment of a communication between the aorta and the pulmonary artery, only a small quantity of aortic blood flowed into the pulmonary artery.

ANEURISM AND SCLEROSIS OF THE PULMONARY ARTERY

Aneurisms of the pulmonary artery are exceedingly rare. They are caused by the atheromatous process within the artery, and also in severe mitral stenosis in consequence of the increased pressure in the pulmonary arterial system. If there is pulsation in the second left intercostal space, with corresponding dullness and absence of the second sound pointing to an affection of the pulmonary valve, and especially the presence of diastolic murmurs, the possibility of an aneurism of the pulmonary artery should be considered. In a case of aneurism of the pulmonary artery observed by Skoda, which during life presented itself under the mask of a mitral insufficiency, there was increased cardiac dullness, systolic murmur over the left ventricle which was transmitted towards the right ventricle and the base of the heart, weak cardiac impulse, diminished diuresis and dropsy. The pulmonary sounds were scarcely audible and there was dyspnœa and marked cyanosis.

In sclerosis of the pulmonary artery even without

aneurismal dilatation there was, according to the case published by Klob and Romberg, during life considerable cyanosis with hypertrophy of the right ventricle, systolic murmur at the apex, accentuated second pulmonary sound, and small, soft, radial pulse. The remarkable feature of these cases is the extraordinarily deep cyanosis with a complete absence of edema until death.

IV

DYSPNŒA AND CYANOSIS IN NEUROSES OF THE HEART

CARDIAC ASTHMA

Dyspnœa during bodily overexertion, especially muscular, is materially different from those attacks of paroxysmal dyspnœa which occur suddenly and without previous warning in individuals who never were dyspnœic before and which subside just as suddenly. Strictly speaking, these sudden paroxysms of dyspnœa should not be looked upon as an exaggeration of difficulties of breathing already existing in cardiac lesions, as they form a symptom-complex of their own, similar to bronchial asthma. Hofmann is correct in placing cardiac asthma in a line parallel with stenocardia. Cardiac asthma, like stenocardia, can be regarded as a neurosis of the cardiac nerve. To explain this polymorphism of stenocardic paroxysms and their radiating phenomena we have assumed the existence of a circle which connects the sympathetic ganglia by their communicating branches with the spinal cord and in which both primary and reflex processes may take place. For a stenocardic attack to be produced, it makes no difference according to our theory whether the causative irritation originates in the end apparatus, *i. e.*, in the sensitive organs of the

coronary arteries, in the transit stations, *i. e.*, the cardiac nerves, in the communicating branch, or in the central apparatus in the spinal cord. Cardiac asthma must be regarded in the same way as a neurosis of the pulmonary or cardiac plexus, but the tracts, whose irritation produces dyspnoea, are different from those active in stenocardia. The irritation of the centrifugal tract may be answered by vascular constriction or dilatation in the lungs and bronchi, so that the oppression and agony find an explanation in an implication of the centripetal tracts of the pneumogastric causing an excitation of the spinal cord and the medulla oblongata. Perhaps there are quite distinct groups of cardiac ganglia which form the end apparatus of this circle, the excitation of which is able by purely anatomical processes to disturb the equilibrium of the heart; in other cases also, there are reflex irritations of the central nervous system which contribute to the production of the asthmatic shock.

PAROXYSMAL TACHYCARDIA

The characteristic stigmata of cardiac asthma, its sudden appearance and equally sudden disappearance, are characteristic of another neurosis, namely, paroxysmal tachycardia. In the pure, uncomplicated cases of paroxysmal tachycardia, respiration is, as a rule, undisturbed. The complexion is more pale than cyanotic, and the respiratory quiet together with the negative pulmonary findings form a striking contrast to the stormy action of the heart. In protracted parox-

ysms, however, cardiac inefficiency, congestion of the lungs, dyspnœa, cyanosis, congestion of the liver, ascites, and anasarca may develop as a sequel to dilatation of the heart. If, however, paroxysmal tachycardia occurs in individuals whose hearts were not normal prior to the attack, as, for instance, in mitral stenosis, myocarditis or pericardial adhesions, then the manifestations of cardiac inefficiency with dyspnœa and deep cyanosis may occur at an early stage.

V

DYSPNŒA AND CYANOSIS IN DISEASES OF THE GASTRO-INTESTINAL TRACT

DISEASES OF THE STOMACH

Special attention should also be given to the manifestations on the part of the stomach and intestine. In some cases of masked forms of angina pectoris with radiation into the esophagus, esophagismus and dysphagia, vomiting, pains in the epigastrium, difficulty of breathing and oppression, occupy the chief place. In another case it is the epigastralgia due to the congestion of liver and stomach, which accompanies the dyspnœa. This is the result of the inefficiency of the right ventricle in cyanotic patients and gives a strange aspect to the general picture.

DISEASES OF THE LIVER AND GALL BLADDER

Dyspnœa from the toxic products of metabolism, which should be rendered harmless by the liver and kidney, offers great difficulty. The clinical picture of toxic dyspnœa due to inefficiency of the renal function, recalls uremia. Not less important for rendering harmless the toxins is the liver. Dogs with Eck's fistula according to several observations become dyspnœic and refuse to eat meat, which permits the inference that alimentary intestinal toxins are capable, after ex-

clusion of the action of the liver, of exciting the center of respiration. In the pathology of dyspnœa in the human subject the liver also seems to play a part, especially when there is renal inefficiency. The cases in which insufficiency of hepatic function through deficient action on the intestinal toxins might be held responsible for the origin of dyspnœa are hinted at in the literature, but insufficiently observed and described. And yet it would be important to go more thoroughly into a question which is full of value both for theory and practice. In all cases of dyspnœa, the character of which points to a toxic causation, a thorough examination of the hepatic and renal functions should be made by modern methods of investigation.

From a clinical point of view, those cases are of special importance in which cardio-pulmonary disturbances are present in affections of the stomach, intestines and gallducts, as dilatation of the right heart, gallop rhythm with maximum intensity behind the sternum and in many cases accentuation of the second pulmonic sound. Such cases have been observed and described by Potain. It is a question whether in these cases we have to deal with a reflex spasm of the pulmonary vessels with increase of pressure and secondary inefficiency of the right ventricle, as occurs in gallstone colics, or with a toxic influence upon the vasomotors of the lungs, exercising a similar effect upon the right heart. In any case, the forms of dyspnœa in which there is simultaneous disturbance of digestion, such as vomiting, diarrhea, dilatation of

the stomach, pyrosis, claim especial interest. It is possible that the poisons developed in the diseased gastro-intestinal tract exert a specific action upon the vasomotors of the lungs and lead to a secondary elevation of the blood pressure in the pulmonary vessels, an assumption somewhat strengthened by a few experiments on animals. An excellent example of the relations between the liver and lungs and the heart are those asthmatic paroxysms, frequently associated with agonizing cardiac arrhythmia of the type of extra-systole, which occur with an attack of gallstones and usually disappear immediately after a gallstone colic and the passage of the stone, without leaving any evidence of trouble behind. From these examples it is evident that in practice there are several forms of cardiac dyspnoea and that frequently in one and the same individual several components may be active, which accounts for the enormous difficulties encountered in both the clinical analysis and in the therapy to be selected.

VI

DYSPNŒA AND CYANOSIS IN INFECTIOUS DISEASES

The cause of dyspnœa and cyanosis occurring in the course of infectious diseases lies in their intimate connection with the pathogenesis of these affections. The causes of infection may lead to pathological changes in the lungs and heart through local, regional and metastatic channels as is the case with most bacteria, or to their multiplication in the blood, as in anthrax. They may act as deoxygenizers and thereby interfere with the interchange of gases, or they may manifest their action by toxins, which possess a chemical affinity for organic cells susceptible to poison, and to which they attach themselves. The toxins of tetanus and botulism select certain neurons of the nervous system, the diphtheria toxin acts on the nerve fibers of certain areas, also upon the heart and the vessels. Both toxins and endotoxins (which result from the decay of the bacterial bodies) may produce distant effects through the circulation as a polyneuritis or grave obstruction to respiration and circulation by the toxic degeneration of various organs (kidney, liver, heart, etc.). Influenza and cholera furnish proofs of both. Secondary infections of the organs injured by toxins contribute their share in intensifying and complicating

the local change of tissue and their distant effect. The hemolytic effect, too, of some bacteria is also to be taken into account as a cause of toxic injury, as is well known in the case of staphylococci and streptococci.

DIPHTHERIA

The chief factor in the causation of dyspnoea in diphtheria is the localization of the process in the larynx or trachea and in descending diphtheria, the involvement of the bronchi. If the pulmonary parenchyma is involved in the way of multiple bronchopneumonic foci, the manifestations will relatively increase. Aside from the localization in the respiratory apparatus, other factors are to be considered in the development of threatening symptoms, namely cardiac weakness caused by degeneration of the muscular substance, hemorrhage in the myocardium, changes in the pneumogastric nerve including its motor nucleus, and also vaso-paralysis with progressively lessening blood pressure. These changes are attributed in the acute stages to the deleterious effect of the diphtheria toxin, while the late paralyzes, in which degeneration of the peripheral nerves manifests itself, are assumed to be due to the action of toxins. In diphtheria which runs its course, as a general intoxication there may be tachypnoea up to sixty a minute, which is usually a premortal sign.

MEASLES, SCARLET FEVER AND VARIOLA

The occurrence of dyspnoea in measles has already been described. In scarlet fever, affections of the air-

tract and lungs are less frequent than in measles. Dyspnœa and cyanosis are observed in malignant forms of scarlet fever and occur as the result of several factors, as complications with pleuritis, endocarditis, pericarditis and myocarditis, and especially in the acute stage of hemorrhagic scarlet fever and in complications with diphtheria; in the later stage with dropsy, hydrothorax and edema of the glottis consequent upon secondary nephritis. Swelling of the face develops from involvement of the cervical lymphatic glands and the cervical cellular tissue, and cyanosis occurs with considerable increase of respiration, affecting speech and deglutition. In a similar way dyspnœa and cyanosis in variola may be occasioned by localization of the affection in the respiratory apparatus; by an affection of the larynx, edema of the glottis, or by extension of the variola to the large bronchi; also by hemorrhagic exudates in the lungs, pleural cavities, pericardium or mediastinum, and finally by injury to the circulatory apparatus. The skin assumes a bluish black coloration from the cyanosis and from the hemorrhages in the derma (smallpox). Dyspnœa may exist in these cases and without any change in the respiratory or circulatory systems.

MALARIA

In malaria, during the stage of chill, there may be considerable dyspnœa with coughing, oppression, precordial fear, even though the circulation is not materially impaired, but it disappears together with the fever. These respiratory disorders, which are accom-

panied by profound cyanosis, are usually caused by hyperemia of the lungs and bronchi. Intermittent dyspnœa occurs in the pernicious forms of malaria which are described as malarial pneumonia and malarial pleuritis. Rare complications are endocarditis and rupture of the heart.

TYPHOID, TYPHUS, RELAPSING FEVER

Dyspnœa may be occasioned by manifold causes in typhoid. The increased frequency of respiration may be of febrile origin. Difficulties of respiration may be the result of obstruction of the nostrils, while grave dyspnœa is caused by ulcerative and perichondritic processes of the larynx, laryngeal edema and croup of the respiratory tract. In extensive multiple atelectasis, in hypostasis and pneumonic affections, in edema of the lungs, in hemorrhages of the lungs and pleuritis, dyspnœa with increased frequency of respiration and cyanosis are easily explainable. An intense dyspnœa occurs in central swelling of the bronchial glands with hemorrhages into the substance and into the surrounding cellular tissue, and it is often, especially in children, out of proportion to the catarrhal manifestations of the lungs (pneumotyphoid). Myocarditis, endocarditis, and, in rare cases, pericarditis, may cause these same symptoms. A high degree of meteorism may influence respiration and circulation by pushing up the diaphragm, and embolism of the pulmonary artery may lead to the well-known and already detailed picture.

There is frequently dyspnœa and cyanosis in typhus

fever owing to cardiac weakness and to the changes in the respiratory tract,—bronchitis, pneumonia, and sometimes edema of the glottis.

In relapsing fever, severe dyspnœa may be present with exceedingly high frequency of the pulse, fear and prostration during the acme of the disease, and shortly before the remission with negative findings in the respiratory organs, and it may disappear rapidly with the remission. When complicated with endocarditis and pneumonia, dyspnœa and cyanosis may occur in the same manner as in typhoid.

ERYSIPELAS, INFLUENZA

In erysipelas of the skin as well as in internal erysipelas, dyspnœa and cyanosis occur from localization of the affection in the respiratory area. Exacerbation of the existing toxo-infectious manifestations and the appearance of dyspnœa generally indicate pneumonia, which usually takes a rapid course and causes death with signs of asphyxia and coma.

There are conditions of suffocation with cyanosis, in influenza, sometimes without any corresponding, objectively demonstrable changes in the lung. They are attributed to functional disturbances in the area of the pneumogastric, or to congestion of the lungs. Dyspnœa and cyanosis may appear at an early stage of bronchopneumonia, which results from the confluence of multiple bronchopneumonic foci, with grave general symptoms, prostration and great frequency of pulse, and they sometimes bear no proportion to the

extent of the pulmonary infiltration. The same was observed in Finkler's contagious streptococcus pneumonia which appeared as a house-epidemic and was similar to influenza. Marked dyspnœa with tachypnœa and small, weak pulse stood in the foreground of the clinical picture.

PLAGUE

In the plague, dyspnœa and cyanosis occur from various causes. Rapidly developing cervical buboes may cause asphyxiation, and the swelling of the lymphatic glands in the thoracic cavity may produce dyspnœa with oppression and an annoying dry cough. Both primary and secondary plague-pneumonia, resulting from ulcerated tonsillary buboes, are accompanied by bloody expectoration containing many plague bacilli; here dyspnœa and cyanosis occur at an early stage and are extreme.

ANTHRAX

Cyanosis with intense dyspnœa, fever, viscid and frequently bloody expectoration, which may have the appearance of prune juice, with contractile pains in the chest, intensified by movement, with a sensation of fear and cold in the internal organs, not infrequently with full consciousness until death, occur in those forms of anthrax in which the lungs and pleura are especially infected with numerous foci of bacilli. The cause of the cyanosis in these cases is not connected with cardiac inefficiency, since the heart, in contrast to other infectious diseases, does not as a rule undergo

fatty degeneration in anthrax, but is usually found to be healthy. Simultaneous intestinal manifestations (anthrax intestinalis) and the demonstration of the bacilli in the blood aid in the diagnosis. The polymorphism of the pathological picture, which depends chiefly on the accumulation of bacilli in the various organs, makes it improbable that the anthrax bacilli produce a specific toxin which attacks vital organs, as is the case, for instance, in diphtheria. Hoffa and Martin have, however, obtained a poisonous substance from pure cultures which in animals produced grave general manifestations and also enlargement of the spleen. The cause of cyanosis in anthrax is supposed to be due to the accumulation of bacilli in the capillary blood (capillary thrombosis), and the subtraction of oxygen from the blood by the bacilli.

MALIGNANT EDEMA

Similarly to primary and secondary pulmonary anthrax, which emanates from a local skin affection, cyanosis with difficulty in breathing and loss of strength occurs in the "Hadernkrankheit," which is caused by the inhalation from "Hadern," or old rags, and is found in persons working in paper factories. This infectious disease is accompanied by headache, sensation of oppression, spastic cough with viscid expectoration, under the picture of a lobar pneumonia. Thoracic pain, however, is frequently absent, the temperature remains low, the sensorium is unimpaired, while the physical manifestations in the lungs are relatively

slight. Although the "Hadernkrankheit" is caused in many cases by the inhalation of "Hadern" dust, containing anthrax spores, in other cases, described by Kranhals, it is not the anthrax bacillus, but the bacillus of malignant edema which causes this infectious disease. The edema bacillus, having once entered the lungs, spreads into the connective tissue of the pleural, mediastinal, pericardial and bronchial glands in the same way as it does into subcutaneous connective tissue. In other cases there is probably a mixed infection of anthrax and malignant edema, perhaps also of other forms of septicemia, which would sufficiently explain the multiformity of the clinical symptoms.

GLANDERS

In glanders, dyspnoea and cyanosis depend upon the localization of the process in the respiratory and circulatory tracts. If the affection is localized in the respiratory tract, especially in the lungs, difficulty in breathing is frequent. This may be intensified in the further course of the disease to pronounced dyspnoea with considerable cough and abundant expectoration, often thin and serous, later more purulent, sometimes bloody and putrid, in consistency similar to nasal secretion. Localization of abscesses in the respiratory muscles and in the heart are likewise of great importance.

TRICHINOSIS

Cyanosis and dyspnoea also occur in trichinosis, if the parts invaded are the diaphragm and the thoracic

muscles. When the trichinæ were chiefly located in the muscular substance of the diaphragm, Traube observed cyanosis with a costal type of respiration, oppression of the chest and a very frequent pulse. If the trichinosis is situated in the respiratory muscles, dyspnœa, with difficult inspiration and extraordinary acceleration of respiration, may assume the character of asthmatic paroxysms, which not infrequently are the cause of death. High fever, profuse perspiration with miliary eruptions, rigidity of the muscles of the entire body, which are sensitive to pressure, contractions of almost all articulations, turgescence of the face and edema of the eyelids, also the eosinophilia of the blood characteristic of trichinosis, may be mentioned as noteworthy signs of the disease. Cyanosis and dyspnœa caused by affections of the respiratory muscles, may be intensified by disseminated bronchopneumonia which frequently accompanies trichinosis, and is often occasioned by occlusion of the smallest branches of the pulmonary artery.

ASIATIC CHOLERA, PERITONITIS

In Asiatic cholera, true dyspnœa is a very rare occurrence, not taking into account the feeling of oppression and fear, palpitation and pressure in the cardiac region. Secondary affections of the respiratory organs, like bronchitis and pneumonia, are not frequent even in the typhoid state of cholera. On the other hand, cyanosis of the lips, ears and extremities, with small or no pulse, is the dominating symptom in the

asphyctic stage of cholera. The lungs become anemic in consequence of the weakness of the right heart, the arteries are empty. The veins are filled with black blood, which, in consequence of the decrease of the *vis a tergo* from weakness of the left ventricle, is tarlike, thick from loss of water, incapable of circulation, and does not become red when exposed to the air. In spite of the increasing viscosity and venosity of the blood and notwithstanding the impairment of the pulmonary circulation, there is no reaction to respiratory irritation. The recurrence of cyanosis with coldness of the extremities in the reactionary stage of cholera (cholera typhoid) is a sign of an exacerbation which not infrequently leads to death after a short period of apparent amelioration.

While dyspnœa practically never occurs in cholera, difficulty in breathing may develop in the course of cholera-typhoid from local inflammatory affections of the respiratory organs (laryngeal and bronchial affections, pneumonic infarcts, edema of the lungs). In isolated cases of cholera-nephritis, there may even be violent dyspnœa which reminds one of uremia. In cholera the moist skin is never absent, but when this is pronounced and there is cyanosis, it is a sign of the worst possible import, as is the presence of dyspnœa, which, however, occurs only in rare cases. The same is true of cholera nostras, except that in the majority of cases cyanosis is not so marked, the skin is somewhat paler and the mucous membranes livid. In severe cases of dysentery, lowered temperature and

dryness and cyanosis of the skin and mucous membranes are prognostically unfavorable signs. Many cases of dysentery complicated with peritonitis and gastro-enteritis present the complex picture of cholera asphyctica,—diarrhea and vomiting,—associated with coldness of the extremities and pulselessness.

The final phase of general peritonitis is likewise characterized by superficial and accelerated respiration, singultus, vomiting, change in the facial contour with cyanosis of the nose, ears and extremities.

VII

DYSPNŒA AND CYANOSIS DUE TO POISONS

In cases of poisoning several factors come into consideration to explain the dyspnœa and cyanosis as injury to the respiratory and circulatory tracts, and blood changes which render the blood less fit or completely unfit for the interchange of gases. The respiratory apparatus may be affected also in various ways: the center for respiration may be excited or paralyzed, the peripheral respiratory muscles and the peripheral nerves, especially their intramuscular terminations involved, or there may be secondary changes in the respiratory tract.

MORPHIN, CHLOROFORM, ALCOHOL, TOBACCO

The chief poison affecting the center for respiration is morphin, which reduces its excitability and finally paralyzes it, so that even an intense venosity of the blood is no longer capable of producing respiratory movements. In poisoning with morphin, therefore, the respiration becomes slow and irregular, accompanied by râles, and the skin is livid or a dark cyanotic hue.

Chloroform has a similar effect; first it paralyzes

the cerebral cortex and then the center for respiration. In the third stage of chloroform poisoning the surgeon quickly recognizes the following manifestations: syncope, asphyxia, stoppage of respiration with cyanosis and dilated pupils, cessation of pulse and of hemorrhage during operation. In alcoholic intoxications there is sopor, general anesthesia, deep stertorous respiration, small, frequent, soft pulse, cyanosis of the skin and mucous membranes, coolness of the extremities. In the gravest cases there is dilatation of the pupils without pupillary reaction, in the less severe cases they are contracted. Similar conditions may prevail in poisoning with ether and laughing gas.

In acute tobacco poisoning, there is vomiting, vertigo, sweating, spasms, palpitation, rapid and irregular pulse, deep dyspnœa with stertorous breathing, edema of the lungs and cessation of breathing. The picture of solanin poisoning is similar.

BELLADONA, HYOSCYAMUS, CALABAR BEAN,
PHYSOSTIGMIN, ACONITE, COLCHICUM

In the multiform picture of poisoning with belladonna and hyoscyamus, dyspnœa and cyanosis occur only as terminal manifestations, occasioned by paralysis of the center of respiration and of the heart. Also intoxications with calabar bean and physostigmin, which are accompanied by contraction of the pupils, may cause dyspnœa with diminishing frequency and finally paralysis of respiration, in addition to manifestations on the part of the intestinal tract. A sim-

ilar picture with grave dyspnœa and asphyctic spasms is presented by aconite poisoning, for which dilatation of the pupils is characteristic. Colchicum intoxications recall the picture of cholera or of arsenical poisoning, and here cyanosis with small, scarcely perceptible pulse, coldness of the extremities with complete consciousness occupy the foreground.

STRYCHNIN, CAMPHOR, CURARE, CONIUM

The principal representative of the poisons which excite the respiratory motor nerves is strychnin. In the course of strychnin intoxication there is considerable dyspnœa with cyanosis and edema of the face with prominent eyeballs in consequence of the tonic-clonic spasm of the respiratory and cervical muscles and the inspiratory immobilization of the thorax. Both dyspnœa and anxiety may appear before the tetanic paroxysms. Dyspnœa, due to spasm of the respiratory muscles, with convulsions, unconsciousness, vomiting, profuse perspiration, small and frequent pulse and lowering of the body temperature have been observed in camphor poisoning.

In poisons, paralyzing the motor terminals of the thoracic and diaphragmatic nerves, as, for instance, curare, asphyxia is easily explainable by cessation of respiration. Cases of curare poisoning have been described in which death ensued within a few minutes without spasms, because the poison paralyzed at once the nerve terminals of the musculature of both trunk

and extremities. In cases where the trunk muscles are paralyzed after the thoracic muscles, there may be general convulsions from the effect of the asphyxiated blood. Such seems to be the case in conium poisoning, in which there is vertigo, stupor, weakness of the heart and of the muscles in conjunction with twitching of the extremities. Respiration becomes labored and retarded, there is dyspnœa and finally cessation of respiration. An ascending paralysis of the spinal cord might have existed in the case of Socrates when he drained the poisoned cup, because he was able to speak and bid farewell to his friends although the muscles of motion and sensation were paralyzed.

TETRODEN FISH, FUNGUS, AGARIC GNAT

This same group of poisons, which paralyzes the muscles of respiration, also includes the poison of the Japanese tetroden fish, which is used for suicidal and homicidal purposes and on shipboard has been the cause of wholesale sickness with fatal termination. Fugu poison, which is prepared from tetroden ova, shows properties similar to curare, but the picture of the poisoning after the ingestion of these poisonous fish varies according to the variety of the species, some cases resembling chloral and opium poisoning, and others, belladonna poisoning or botulism, in which mydriasis, ptosis, considerable paroxysmal dyspnœa with dryness in the throat, dysphagia and disturbance of vision are present. In fungus poisoning there is,

aside from the gastro-enteric symptoms and mental disturbances as in atropin poisoning, dyspnœa with paroxysmal attacks of suffocation, and stertorous breathing owing to edema of the lungs; in agaric gnat poisoning there is also retardation of the pulse.

BARIUM SALTS, CORROSIVE SUBSTANCES

Intestinal manifestations with slow pulse, dyspnœa, anxiety and coldness of the extremities, convulsions from suffocation and paralysis of the heart occur in intoxication with barium salts. In poisoning with corrosive substances like sulphuric acid, nitric acid, hydrochloric acid, caustic soda and bichlorid of mercury, there are difficulties in breathing, especially in cases where the poison cauterizes the larynx and trachea, so that the air has difficulty in gaining access to the lungs, owing to the considerable swelling of the mucous membranes.

The well-known symptoms of internal poisoning by caustic ammonia, *i. e.*, salivation, vomiting, diarrhea and considerably accelerated respiration may also occur, if the poison has been introduced by the rectum. This happened in a case under my observation, where a patient had used an ammonia enema in mistake for glycerin. There was swelling of the buccal mucous membrane and a secondary ulcerous stomatitis with Vincent's organisms (*spirochetæ* and fusiform bacillus), as seen in mercurial stomatitis.

IRRITATING GASES AND VAPORS

Inhalations of irritating gases and vapors, as chlorine, bromine, iodine, hydrochloric acid, hydrobromic acid, nitric acid, nitrous acid, ammonia, etc., cause disturbance of respiration by reflex spasm of the glottis and constriction of the bronchioles, means of defense, also by secondary changes in the mucous membranes of the larynx and the bronchi, which may lead to edema of the glottis, severe bronchitis and lobar pneumonia. Aside from the local manifestations, these gases may also produce distant effects upon respiration and circulation by causing injury to the heart, to the center for respiration and by direct contamination of the blood.

After the inhalation of a concentrated mixture of carbon dioxide, dyspnoea and unconsciousness may occur, symptoms analogous to those of suffocation.

DIGITALIS, HELLEBORE AND VERATRUM

Digitalis and hellebore should be mentioned among the poisons which cause dyspnoea by direct action upon the cardiac function. Vomiting, violent headache, dimness of vision, chromatopsy, tinnitus aurium, fainting, cold perspiration, marked muscular weakness, with consciousness relatively intact, considerable retardation of pulse, make up the clinical picture of these intoxications. General cyanosis with sopor, coma, and convulsions occur with reduction of the body temperature,

increase of pulse frequency and arrhythmia. There is a similarity between the symptom-complex of digitalis and veratrum poisoning, except that the gastric manifestations with salivation, also the diminution of cardiac function and paralysis of the center for respiration appear more rapidly in veratrum poisoning.

PHOSPHORUS

In phosphorus poisoning, cyanosis due to the engorgement of the venous system is hidden by the synchronous icterus. The labored breathing is explained by the weakening of the cardiac function. In rare cases dyspnoea and cyanosis are observed from a rupture at some point in the aorta where there is fatty degeneration.

CARBON MONOXID

Blood poisons may produce dyspnoea and cyanosis in that they render the blood unfit for respiration, both for the interchange of gases and the transmission of oxygen. This may take place in two ways. Either the poison absorbed assimilates with the hemoglobin without changing the red blood corpuscles, or it effects a change in the oxyhemoglobin of the red blood corpuscles with or without their destruction. Met-hemoglobin is formed in the first combination and is equally unfit for respiration or transmission of oxygen. The best known representative of the first group is carbonmonoxid, which displaces the oxygen from the oxyhemoglobin, assimilates with hemoglobin and forms a firmer combination, carbonmonoxidhemoglo-

bin, which is incapable of either oxydation or respiration. The most prominent symptoms in this intoxication, therefore, are dyspnœa with impending asphyxia, abnormally high frequency of pulse and decreasing blood pressure, sleepiness and diminished reflex excitability with a tendency to inflammatory affections of the lungs. In the soporific stage, respiration may be regular and scarcely affected, or there may be intermissions in the respiration and death follows with or without terminal convulsions. Occasionally clonic and tonic spasms of the extremities may occur early with trismus and facial spasms.

In carbonmonoxid poisoning, the color of the mucous membranes and of the skin is sometimes bright red, at times pale, grayish or livid; frequently there is hyperemia in spots on various parts of the body. Cyanosis and edema occur as terminal symptoms only toward the end, due to the disturbance of circulation. Aside from its effect upon the hemoglobin, carbonmonoxid seems to exert direct toxic effects upon the vessels and the nerve elements, as is proved by the numerous cases of histologically demonstrated affections of the central and peripheral nervous system and of the vessels (disseminated encephalitis, especially of the cerebral ganglia, sometimes immediately following the poisoning). In an eighteen-year-old girl poisoned by illuminating gas and treated at our clinic there was, aside from unconsciousness and labored breathing, edema of the face, hyperemia of the skin in limited areas, small, compressible arrhythmic pulse, pupils

at first markedly contracted and without reaction, elevation of temperature, tetaniform spasms of the upper extremities and pectoral muscles, exaggeration of the patellar reflexes, bilateral Babinski reflex, sugar and lactic acid in the urine. After two days of unconsciousness there was a rather rapid subsidence of all symptoms and recovery.

SUBSTANCES FORMING METHEMOGLOBIN

The second group of poisons is characterized by the formation of methemoglobin. There are substances which change oxyhemoglobin into methemoglobin without destruction of the red blood corpuscles, as amylnitrit, antifebrin, kairin and nitrobenzol. Others form methemoglobin and change the structure of the red blood corpuscles, as the chlorats, nitrits, and anilin. In some poisons the formation of methemoglobin commences at an earlier stage than do the changes in the red blood corpuscles, and the degree of chemical change in hemoglobin is not always proportionate to the morphological injury to the cell body of the red blood corpuscles. Certain poisons dissolve hemoglobin easily, forming methemoglobin without material change of the stroma, as, for instance, sodium nitrit, potassium permanganat, pyrogallie acid, antifebrin. Others again, especially the chlorin salts and arseniated hydrogen, preferably destroy the red blood corpuscles, though not always immediately. The dissolution of the erythrocytes with the transfusion of the lecithin into the plasma, causes a splitting of the lecithin

and the formation of glycerophosphoric acid which acidulates the blood. In other cases, there is a toxic formation of acids by inhibition of the oxydation of the acids due to the albuminoid metamorphosis of the tissues, as lactic acid, the presence of which in the blood in a case of arsenical poisoning was demonstrated by H. Meyer. Both the increase of the acidity of the blood and the formation of toxic acids may in such cases of poisoning play a rôle in the production of dyspnœa, inasmuch as in acid intoxication the tissues lose their ability of absorbing the oxygen which the blood offers them.

ANTIPYRIN, BENZOL DERIVATIVES—ACETANILID

Cyanosis after the use of antipyrin has frequently been observed. Bamberger reported the case of a lady who, each time after the administration of fifteen grains of antipyrin for the relief of migraine, became blue without any other symptom. I personally observed a case of hysteria in which there was considerable cyanosis of the skin after the administration of ten powders of seven and a half grains each of antipyrin. The face was especially cyanotic and the pulse was accelerated, with a condition bordering on collapse and with hysterical contractions of the extremities. The treatment with antipyretics—antipyrin, lactophenin, etc.—to cure febrile conditions may be followed by cyanosis, which, however, does not have the disquieting significance, especially in typhoid, which accompanies other disturbances of the circulation.

In anilin poisoning as well as in poisoning with benzol derivatives, especially acetanilid, there is in addition to the vertigo, headache, dyspnœa, muscular weakness with fibrillary contractions and somnolence, also cyanosis as the most prominent symptom. It is frequently accompanied by hematuria, strangury and oftentimes by icterus. Poisoning with nitro-compounds of the aromatic series (nitrobenzol, dinitrobenzol, nitrotoluol), which are employed in blasting operations, produces considerable cyanosis which sometimes appears even before the other symptoms of poisoning. The blood is of a chocolate color, but does not always contain methemoglobin, according to the experiments of Huber and Haldane, but rather, as it would seem, a nitro-compound of hemoglobin, which is also incapable of respiration (Dehio: anilin black). In workmen employed in rectifying petroleum, Eulenberg frequently observed cyanosis of the lips and a bluish complexion.

NITROGLYCERIN, AMYL NITRIT AND CARBOLIC ACID

Nitroglycerin and also amyl nitrit poisoning cause considerable cyanosis, severe headache, dyspnœa and muscular debility, and the cyanosis outlasts all the other manifestations even in favorable cases. In the three fatal cases of nitroglycerin poisoning observed by Nyström, death ensued, without convulsions, in coma after considerable dyspnœa and cyanosis. Carbolic acid poisoning also causes dyspnœic breathing with cold, cyanotic skin as a prominent symptom, in addi-

tion to the well-known corrosive effect on the esophagus and the stomach, the dark olive-colored urine and the loss of consciousness.

ARSENIATED HYDROGEN AND POTASSIUM CHLORAT

In poisoning with arseniated hydrogen and potassium chlorat, cyanosis and dyspnœa form a prominent symptom as a consequence of the serious injury to the blood, aside from the other well-known manifestations of intoxication. A not uninteresting observation in these cases is the appearance of nuclear erythrocytes in the blood, occasioned by toxic irritation of the marrow. A working man, observed by me, who had been employed in filling a balloon, inhaled arseniated hydrogen and developed sleepiness and considerable cyanosis. The blood contained large quantities of nuclear erythrocytes without any eosinophile cells and without myelocytes, a fact which under certain circumstances may have a diagnostic significance as against other intoxications, especially cholera.

Another factor deserves mention in these cases of poisoning. The destruction of the red blood corpuscles, which takes place as methemoglobin is being formed, may lead to obstruction of the vessels in the internal organs, especially the lungs, by the débris of the destroyed erythrocytes and in this way cause disturbances of the circulation and respiration. A similar process occurs in burns of the skin, in which there are cyanosis, dyspnœa, coma and low temperature with lessened blood pressure, so that not only the toxic ef-

fect through resorption of the poisons formed at the injured places should be considered, but also multiple thrombi due to the débris of destroyed red blood corpuscles.

NITRITS—SODIUM AND POTASSIUM

The nitrits have a special significance in the pathogenesis of cyanosis. It is a well-known fact that both sodium and potassium nitrit belong to the poisons which form methemoglobin. The nitrats have no such effect, but it is possible that they are changed into nitrits in the organism. Since the bacilli of putrefaction and pathogenic fungi, as anthrax bacilli, may reduce nitrats to nitrits, a similar change may well take place in the intestinal canal and perhaps also in the blood. Nitrits cause cyanosis by the formation of methemoglobin, which paralyzes the muscles of the blood vessels and reduces the blood pressure. Even after medicinal doses of sodium nitrit (six grains) cyanosis has been observed.

In 1893 Emmerich and Tsuboi tried to explain Asiatic cholera as a nitrit poisoning, based upon the discovery of methemoglobin in the bodies of guinea pigs which had died of cholera, and upon similar findings in the blood of test animals, poisoned with sodium nitrit. It had been known for a considerable time that cholera bacilli are capable of oxydizing ammonia from its combinations into nitrous acid and that they are distinguished from many other bacteria in being able to change nitrats into nitrits. During the chol-

era epidemic in the autumn of 1892 I paid special attention to the nitrifying ability of the cholera bacillus, examining not only the cultures of the cholera bacillus, but also the feces of patients. The gelatine cultures of the cholera bacillus obtained from the stool of the first fatal case in Vienna, were stained red (nitrous acid) by both sulfanilic acid and naphthylamin, while with diphenylamin they promptly reacted to nitrats. On the other hand, I have not succeeded in demonstrating either nitric or nitrous acid in the feces of cholera patients, dead or alive, after they have been acidulated with sulphuric acid. Examinations of the blood and urine of cholera patients for the presence of nitrits have not, to my knowledge, been made, while personally I did not have an opportunity of making them. Besides, the picture of nitrit poisoning in the human being does not correspond at all with that of cholera.

The usual manifestations in nitrit poisoning consist of headache, great weakness, nausea and cyanosis. Isolated cases of poisoning with large doses of nitrit have been known, where there were grave manifestations of collapse and simultaneously vomiting and diarrhea. This may easily happen if part of the ingested nitrits has already undergone chemical decomposition in the stomach, and liberated nitrous acid. True gastro-enteritis with uncontrollable vomiting and ricewater stools has not been observed in these cases. Although at the present time nobody harbors the idea of connecting single symptoms of cholera intoxication

as cyanosis with nitrits, yet in recent times intestinal autointoxication has been observed in which nitrits probably formed in the intestines under the influence of nitrifying bacteria, seemed to have played a part.

Hymans van den Bergh and Grutterink found in many cases of enterogenous cyanosis from methemoglobinemia, nitrits showing Griess's reaction in the blood. With strict milk-diet the cyanosis disappeared after twenty-four hours, but reappeared upon the resumption of a mixed diet. Gibson and Douglas observed in the blood of a cyanotic woman of thirty-six, complaining of vertigo, headache and diarrhea, methemoglobin and nitrits, the latter also in the saliva. The bacteriological examination of the blood revealed the presence of a bacterium of the colon group, to which the formation of nitrits and methemoglobin was attributed.

NITROUS ACID, PRUSSIC ACID

In poisoning by the ingestion of nitrous acid there are corrosive effects, asthmatic attacks of suffocation and asphyxia with severe cyanosis. On the other hand it has been observed that the intoxication by this acid produced almost the identical symptoms as did the poisoning by ammonia.

Poisoning with prussic acid leads rapidly to death, either by instantaneous general paralysis or by stupor, paralysis of the heart and convulsions, accompanied by an initial cry as in epileptic seizures. In chronic intoxications with substances which yield prussic acid,

as bitter almonds, there is in addition to acceleration of the respiration and oppression of the chest, dilatation of the pupils, salivation, coldness of the extremities and cyanosis of the mucous membranes, the face and the entire body.

Robertson and Winne reported seven cases of prussic acid poisoning after the ingestion of the beans of the *faseolus lunatus*. Four of them terminated fatally within eleven hours and death ensued with severe dyspnœa and convulsions. From first to last the picture was the result of a deep disturbance of the function of respiration.

Dyspnœa in prussic acid poisoning does not depend upon the formation of cyanmethemoglobin, which it produced only in experiments in a solution of methemoglobin. Numerous experiments have demonstrated that the cyanosis is not caused by the blood being unable to give up oxygen, as is the effect in poisoning with carbonmonoxidhemoglobin, but by the fact that the tissues, especially the ganglionic cells of the center for respiration, lose their power of absorbing the oxygen, which is abundantly offered to them. The deficiency of oxygen resulting therefrom leads to a most violent excitation of the center for respiration which rapidly passes into paralysis. On account of its strong effect upon the center for respiration prussic acid has been proposed as an antidote to chloroform poisoning, but so far it has been employed only in veterinary practice. Horday used it with good success on a large number of dogs.

SULFURETTED HYDROGEN

The effect of sulfuretted hydrogen is similar to that of prussic acid. In light cases of poisoning there is only irritation of the conjunctiva and nasal mucous membrane, but at times also vertigo, nausea and severe dyspnœa. If large quantities of the poisonous gas invade the respiratory tract, as may happen to workmen in sewers and drains, respiration is at once accelerated, soon becomes labored and highly dyspnoëic, the pulse weak, the skin cold and cyanotic, and the abdomen exceedingly distended. There may be convulsions and coma, followed by death from edema of the lungs and cessation of respiration. The asphyctic manifestations may subside even in grave cases and recovery takes place, different from carbonmonoxid poisoning. This is explained by the rapid exhalation of the poison from the lungs, and is corroborated by the fact that the expired air in man and animals, poisoned with sulfuretted hydrogen, blackens a sheet of acetate of lead paper held before the mouth. It also agrees with the fact that in intravenous injections, sulfuretted hydrogen is not absorbed by the arterial blood. This rapid elimination of the poison explains too why the intoxications which happen to chemists, and the autointoxications due to the absorption of the intestinal sulfuretted hydrogen into the circulation, do not usually take a dangerous course. In the rectal application of sulfuretted hydrogen, recommended by Altmann for the treatment of tuberculosis, no toxic

symptoms—dyspnœa or cyanosis—were observed. On the other hand, Hyman van den Bergh and Gutterink have reported cases of enterogenic cyanosis with obstinate constipation, in which the blood is said to have contained sulfhemoglobin, which disappeared after evacuation of the intestine. This result is almost unexplainable, since all attempts to demonstrate during life sulfhemoglobin in the blood, even in grave and fatal intoxications with sulfuretted hydrogen, have been unsuccessful.

BOTULISM, TETANUS TOXIN, POISONOUS FISH

Botulism, that is, poisoning by decomposed sausages and albuminous food (both meat and vegetable preserves), resembles the symptom-complex of atropin poisoning, in so far as the intoxication with the toxin of botulism, but not when there is an infection with the bacterium coli and Gaertner's bacillus. Among the symptoms are dyspnœa with precordial anxiety, annoying and often spasmodic cough, dysphagia, glossoplegia, dryness of the mouth, paresis of accommodation, paralysis of the ocular muscles, ptosis with cardiac weakness, cold, pale skin, and lividity of the mucous membranes. These bulbar symptoms result from the combination of the toxin of botulism with the ganglionic cells of the bulbar nuclei.

A similar elective affinity for the nervous system, especially for the ganglionic cells of the anterior cornua and of the bulbar nuclei, is characteristic of tetanus toxin. The symptoms, which appear at the

climax of an attack, are dyspnœa with fear, livid skin, frothing at the mouth due to spasm of the muscles of the glottis, tongue and respiration.

Intoxications from eating poisonous fish (unsalted sturgeon) also belong to this group. These fish contain a ptomain, with an action similar to atropin, which is destroyed by boiling and is thus different from the poison by the Japanese tetroden, which is not destroyed by heat. The symptoms after the ingestion of the various species, sphyrena, coryphen and scomber, are a red or violet complexion, often considerable edema, especially of the lips and eyelids, difficulty in breathing of an asthmatic nature, occasionally acute coryza with sneezing, lachrymation and spastic cough, and resemble closely urticaria of the skin and mucous membranes, after partaking of strawberries, lobster, etc.

INSECT STINGS, SNAKE BITES

Insect stings may occasion general symptoms with fainting, cardiac weakness, difficulty in breathing, cyanosis of the lips, coldness of the extremities and fatal collapse, especially when the sting empties its poison directly into a blood vessel. Stings in the mouth or fauces from eating fruit in which bees or wasps are hidden, become dangerous on account of the edema of the glottis they may produce (Bollinger). Stings by hornets (*vesta crabro*), whose poison is said to immunize against viper poison according to experiments made by Physalix, may be followed by severe general symptoms, with dyspnœa, sensation of

oppression and collapse. The same is true of scorpion stings.

Finally, rapidly appearing dyspnœa with slow and irregular respiration, facies hippocratica with coma and paralysis, cyanosis and scorbutus-like hemorrhages into all tissues of the body, without injury of the vessels consequent upon hemolysis, are symptoms of the poisoning from snakebites.

BERI-BERI, ANESTHETIC LEPROSY

Beri-beri, a disease occurring in Japan and China, is regarded by some as an intoxication due to decomposed vegetable or animal matter, and by others as an infectious disease. It frequently occurs with dyspnœa and weak cardiac function, paralysis of the lower extremities, atrophy of the muscles, similar to alcoholic neuritis and myxedema of the skin. The dyspnœa is occasioned partly by neuritic changes in the pneumogastric and phrenic nerves, in part by fatty degeneration of the cardiac muscle, especially in those forms running a rapid course and leading to death through cardiac inefficiency with cyanosis.

In anesthetic leprosy, bulbar symptoms have to my knowledge never been described, although neuritic processes belong to the characteristic findings due to leprous infiltration of the peripheral nerves, the ulnar and major auricular nerves. In *lepra tuberosa*, as already mentioned, localization of the disease in the larynx, trachea or even lungs, may cause severe dyspnœa with stenotic symptoms. The susceptibility of

leprous patients to iodine preparations is well known. Frequently they react to comparatively small doses of potassium iodide in a way similar to tuberculin, causing hyperemia and edema of the affected parts. In laryngeal lepra there may be stenosis from edema of the glottis after the administration of potassium iodide.

VIII

DYSPNŒA AND CYANOSIS IN GENERAL DISEASES

BRIGHT'S DISEASE

Dyspnœa and cyanosis occur in subacute or chronic affections of various organs when secondary changes in the circulation and respiration are added. Bright's disease, in which dyspnœa may be due to various causes, belongs especially in this class.

Renal asthma oftentimes occurs acutely and in paroxysms without demonstrable bronchial or pulmonary affection. In other cases there is a transitory bronchial catarrh with râles, which change their site and disappear after the paroxysm. Besides these mobile and transitory edemas there is also a genuine pulmonary edema with characteristic sputa. In the final phase, the dyspnœa is permanent with nightly exacerbations as in cardiac asthma, except that hemoptysis and hemorrhagic infarcts are rare. The hemorrhages from the lungs and the bronchial mucous membranes, if they do occur at all, are as a rule the expression of a hemorrhagic diathesis.

Cyanosis does not develop even though the functional energy of the left ventricle be weakened, provided that the anemia, which in nephritic cases sometimes reaches a high degree, is marked.

CHRONIC NEPHRITIS

There are, however, cases of chronic nephritis in which the blood has suffered little in spite of the chronicity of the affection. In these cases the clinical signs may begin with gallop rhythm, cyanosis, congested lung, enlarged liver, sometimes with reduced diuresis if previously there was polyuria, so that the differential diagnosis as to the presence of *cor renale* or *ren cardiacus* may cause difficulty. The differential diagnosis may depend upon the variations in the excretion of nitrogen, especially of urea, retention changing with considerable excretion, diminution of phosphoric acid as a rule, especially that form combined with alkaline earths, upon the normal relation of sulphuric acid to nitrogen in conjunction with normal or reduced diuresis, increased concentration of the blood, especially in contracted kidney, and other clinical symptoms of primary renal affection, such as retinitis.

CONTRACTED KIDNEY, CYSTIC KIDNEY

Gallop rhythm is a frequent occurrence in genuine contracted kidney, while arrhythmia is rare. In arteriosclerotic contracted kidney, however, cardiac arrhythmia is frequent in consequence of the myocarditic changes of the heart—and there may be hard pulse with high blood pressure during the dyspnœic paroxysms in spite of inefficiency of the ventricle. It should also be remarked that congenital cystic kidneys, unless they are complicated by nephritis, may be entirely free from increased blood pressure, even in

manifest uremia, forming a striking contrast to the uremia of contracted kidney. Lowered or not materially increased blood pressure in the presence of uremic manifestations should therefore remind us of cystic kidney and demands a careful palpation of the renal region. If cystic kidney is kept in mind as a possible cause of uremia, it will not be difficult to establish a positive diagnosis, in case the bilateral, usually non-fluctuating, nephritic enlargement can be felt, which generally makes the characteristic impression of a bunch of grapes. Cystic kidneys complicated with chronic nephritis lead to hypertrophy of the left ventricle and high blood pressure. The fact of uremia and increased blood pressure not always running in parallel lines is perhaps of theoretical importance in looking upon uremia as an intoxication.

RENAL ASTHMA VERSUS CARDIAC ASTHMA

The inefficiency of the hypertrophic left ventricle in the course of latent contracted kidney may occur acutely, and not infrequently the renal asthma is the first symptom of the affection. For the purpose of differential diagnosis from asthmatic paroxysms in primary cardiac affections, it should be noted that the urine voided during the paroxysm is albuminous, light colored, less rich in coloring matter, and larger in quantity than in cardiac asthma. In the latter affection the urine is usually dark, concentrated, contains urobilin, has a high specific gravity and deposits abundant *sedimentum lateritium* after the asthmatic

attacks. The urine in nephritis may remain light yellow even if febrile processes develop in the lungs or pericardium. The urine in Bright's disease does not always show the characteristics of febrile urine, or that of congestion, in spite of the presence of stasis and fever.

LATENT NEPHRITIS

In latent nephritis a simple acute catarrh of the respiratory tract may sometimes suddenly and without warning present the clinical picture of severe dyspnœa and general dropsy with numerous râles, causing death from a complicating edema of the lungs. This is explainable by the fact that in nephritis an irritation of a tissue is frequently provocative of edema. Just as a simple catarrhal laryngitis may produce edema of the larynx, small pneumonic foci may lead to edema of the lungs. In the same way sudden dyspnœa, cyanosis and coma in chronic Bright's disease are not infrequently semiotic signs of commencing pneumonia which may in a very short time lead to edema of the lungs and death, without the objective signs of pneumonia being clearly demonstrable. In such cases constant elevation of temperature may be diagnostically of value in contrast to the purely toxic form of uremic dyspnœa and of uremic coma, in which the temperature of the body generally falls, provided there is no simultaneous cerebral hemorrhage, which would, after originally lowering the temperature, cause a rapid rise.

At times pulmonary edema may occur from bodily overexertion, mountain climbing, alcoholic intoxication, sexual excesses, use of certain drugs, as opium, even in small quantities, also from acute gastric catarrh in consequence of sudden weakness of the left ventricle, while the right ventricle still functionates. In these cases there are also direct clinical signs of disturbed function of the left ventricle, as dilatation and gallop rhythm.

HEMOPTYSIS IN CONTRACTED KIDNEY

In cases where these signs on the part of the ventricle are absent, and yet there are symptoms of stasis in the venous and pulmonary systems with high blood pressure, the cause will be found in degeneration of the muscle of the left auricle. Bamberger in his lectures has referred the hemoptysis, which sometimes occurs as the first warning of a contracted kidney, to a hyperemia of the lungs from dilatation of the left auricle. In the administration of digitalis or ergotin to remove these symptoms, caution should be exercised. On account of the abnormal tension of the aortic system, a cerebral hemorrhage may easily be produced, especially in cases of Bright's disease prone to such a result. The diagnosis of dilatation of an auricle is still further confirmed, if during prolonged observation cyanosis as well as the other manifestations of congestion in the lungs and other organs increase, while the examination of the left ventricle does not reveal any striking changes; neither a progressively increasing

dislocation of the cardiac impulse, nor in a functional respect, a decrease of the arterial tension. The formation of thrombi in the right auricle, secondary emboli in the lung, also of thrombi in the dilated left auricle and left ventricle may impart to the clinical picture considerable polymorphism and cause great difficulty in the diagnosis.

CYANOSIS OF UREMIA

Pronounced cyanosis of an edematous face with clonic spasms and subsequent coma, after preceding headache, vomiting, reduction of the pulse frequency, with frequently but not always diminished diuresis, belong, as is well known, to the picture of an uremic attack and are occasioned by the engorgement of the tissues during the spasm. The cyanosis of the face occurs in the same way as in epilepsy. The paroxysmal dyspnœa of nephritis, which is generally designated as uremic dyspnœa, occurs with or without cyanosis if there are no convulsions, and the cyanosis in these cases is entirely dependent on the behavior of the heart. In uremic dyspnœa cyanosis is entirely absent, if the dyspnœa is occasioned by toxic irritations of the bulbar center for respiration by excrementitious urinary substances.

REDUCED HEMOGLOBIN IN BLOOD

According to Labbé the capillary blood contains 0.5 to 1% of reduced hemoglobin. In valvular defects the quantity of the reduced hemoglobin increases

to 2%, especially after exertions, in disturbances of compensation up to 3%, 5%, and at times even 7%, and in congenital cardiac defects up to 10%. In uremic dyspnœa the quantity of the reduced hemoglobin does not run parallel with the intensity of the dyspnœa. In three cases 2% were found. Labbé concludes from these investigations that uremic dyspnœa is of toxic origin, while cardiac dyspnœa is rather to be attributed to obstruction of the circulation.

DIABETES

Since Kussmaul's classic presentation, the dyspnœa which ushers in coma diabeticum is looked upon as an integral symptom of the pathological picture. This form of coma, characterized by an accelerated, regular, strongly dyspnœic and deep respiration, is designated as coma diabeticum. In true diabetic coma cyanosis is absent in spite of considerable dyspnœa, so that the absence of cyanosis in the presence of inspiratory dyspnœa without orthopnœa is a direct reminder of diabetes. Diabetic dyspnœa is regarded as an acid intoxication on account of the abundant excretion of acetone, di-acetic acid and B-oxybutyric acid. Through the acidulation of the blood a large part of the alkali in the blood is neutralized. Since the alkali of the blood is the important element which chemically binds the carbon dioxid, the decrease of blood alkalinity together with the reduction of the carbon dioxid capacity of the blood in diabetes is intelligible. An increase in the venosity of the blood therefore does not take place

in diabetic acid-dyspnœa. In this way the absence of cyanosis in pure cases of diabetic coma, not complicated by pulmonary and cardiac affections, is explained.

In contrast to diabetic coma, which is introduced and characterized by dyspnœa, there are also cases of fatal coma in diabetes in which Kussmaul's dyspnœa is absent. These cases of non-dyspnœic coma in diabetes have been described by Lossen (of Lichtheim's Clinic in Königsberg) and do not seem to stand in the same relation to the acidosis of diabetes, as effect to cause. Even those forms of diabetic coma where the urine and blood analysis point to an acid intoxication, do not in all cases present the typical picture of Kussmaul's respiration. There are cases in which there is dyspnœa without deepening of the respiration, but with disturbances of the respiratory rhythm, as Cheyne-Stokes' respiration, and again cases where there are epileptiform spasms without real dyspnœa as an expression of the serious damage done to the central nervous system by the abnormal metabolic products in diabetes.

CYANOSIS IN DIABETES

Cyanosis, as mentioned, is absent in diabetic coma, but in the acute exacerbations of diabetes it may appear from complications with organic affections of the respiration and circulation. There are some cases of chronic diabetes with a slight cyanotic reddening of the face, especially of the ears and nose, so that an impression is created that the patient is a alcoholic with a weak heart. These bluish discolorations are

observed both in obese and in lean diabetic patients, affected with afebrile tuberculosis. Therefore, in cyanotic patients, both obese and with afebrile tuberculosis, the urine should be examined for sugar, in order not to overlook the primary affection. It was just this manifestation and the absence of night sweats that caused me to diagnosticate diabetes mellitus as the primary complaint in a phthisical patient who had been sent by his physician to Meran to take a grape cure.

Of a totally different nature are those forms of glycosuria in which in addition to an acute dilatation of the heart cyanosis appears with grave and sometimes fatal forms of tachycardia. Both the glycosuria and tachycardia should be regarded as bulbar symptoms. In especially complicated cases the interpretation of the glycosuria may be facilitated by an investigation of the metabolism, as the latter in all genuine forms discloses an increased secretion of organic and inorganic urinary substances corresponding with the diabetic autophagia.

HYDROPHOBIA

Dyspnœa associated with precordial fear, with frequent sighing and unusually deep inspiration, with a sensation of constriction in the throat is an early symptom of hydrophobia. In its further course the dyspnœa increases until it reaches a true respiratory spasm, and is accompanied by an extreme hyperesthesia, which affects nearly all the nerves, especially the pneumogastric, phrenic, glossopharyngeal and ac-

cessorius. Hydrophobia and aërophobia (fear of water, or of the slightest draught of air), even general convulsions at the sight of water and of a crowd of people, dysphagia, etc., are only part manifestations of the hyperesthesia and increased reflex excitability, which is an expression of the profound damage to the nervous system. There is a striking contrast between the cyanosis and the extreme pallor of the skin, especially of the face, which assumes a livid cyanotic color only during the convulsive attacks which are similar to those occurring in tetanus.

In the paralytic stage, which is characterized by decrease of the dyspnœa and of the reflex excitability, by accelerated and stertorous respiration and increasing cardiac weakness, with small and exceedingly rapid pulse; disturbance of the circulation may cause cyanosis as a premortal sign that may persist until death. Death ensues either under asphyctic convulsions or in quiet coma. The dyspnœa of rabies is of bulbo-medullary origin and its characteristic is the absence of cyanosis in the stage of the excitement, just as is the acid dyspnœa of diabetes. In the course of hydrophobia intense acetonuria, together with dyspnœa, has been noted from the very first in my patients. This symptom may have a diagnostic significance in contradistinction to hysterical hydrophobia. Then too acetonuria is of special theoretical interest in an affection characterized by considerable excitation with complete consciousness, in which coma occurs only as a terminal manifestation, because acetonuria

by way of contrast always accompanies the comatose stage of diabetes. A remarkable fact is the resistance shown by patients with hydrophobia to narcotics, especially to preparations of opium. In one of my patients I injected morphin up to seven grains without giving the slightest relief. Indeed, it appeared to me that the morphin had effected an increase in the convulsions. This power of resistance in this affection to the specific respiratory poisons recall the refractory behavior of cats against the same poison, a fact with which experimental pathologists are thoroughly familiar.

ANEMIA

In cases of grave anemia, either true or post-hemorrhagic, there are no material changes in respiration unless the anemia attains an extreme degree. The process of oxydation shows a perfectly normal behavior in the consumption of oxygen by the tissues, and in the elimination of the carbon dioxid. Highly anemic patients bear impoverishment of the blood from defective circulation of oxygen very well, and avoid everything which might lead to a greater demand for oxygen, including increased activity of the respiratory muscles. Their respiration is shallow and superficial and they do not become dyspnœic during muscular quiet. When the demand for oxygen is increased, as in muscular overexertions, dancing, carrying heavy loads, etc., the supply of oxygen may become insufficient. This insufficiency is shown as polypnœa with the subjective sensation of air-hunger

and pronounced dyspnœa, the purpose of which is to increase the aspiration of the blood into the thorax and to increase the circulation and aëration of the lungs. As muscular activity is of extreme importance in the respiratory interchange of gases, this myogenic dyspnœa in anemia simply means a repetition of the physiological process which occurs in muscular exertions and disappears in muscular quiet. The dyspnœa of anemic patients disappears with rest in bed and is an important factor in the therapy of chlorosis, even in those forms not complicated by a dilatation of the heart. The extra work demanded of the thoracic muscles can be estimated by the dyspnœa from which chlorotic girls suffer who wear tight corsets and experience no further trouble upon the removal of the corsets. All these forms of myogenic dyspnœa are the result of irritation of the center of respiration by the metabolic products of active muscles, and anemia is a condition which develops this physiological irritation more easily and rapidly.

In cases of anemia accompanied by dilatation of the heart and involvement of the muscular substance, the dyspnœa is due to these complications, and the mechanism is the same as that of the cardiac form of dyspnœa.

LEUKEMIA

Dyspnœa may be accentuated in leukemia to the most violent, asthmatic paroxysms, which are due, however, less to the changes in the blood than to a permanent or transitory weakness of the myocar-

dium. Bamberger has emphasized this fact: "Neither the changes in the blood of themselves nor the obstruction to respiration by an enlarged spleen or liver, nor the enlargement of the bronchial glands are a sufficient explanation, because dyspnœa may come and go without any change in these conditions; and, besides, there is no constant relation between them." In doubtful cases the differential diagnosis as to whether dyspnœa is of myogenic, hematogenic or cardiac origin, may be established by enjoining absolute muscular rest and making a therapeutic test with cardiac remedies, as digitalis, strophanthus, ether and camphor.

Similar to leukemia, subjective dyspnœa, especially in exaggerated muscular exertions with negative pulmonary findings, frequently associated with vertigo and faint feeling, is an important semiotic sign of those forms of fatty heart which are at times accompanied by considerable pallor of the skin and mucous membranes. In cases where the blood examination does not disclose qualitative changes, we find in addition to the dilatation of the heart, the cardiac sounds dull and soft, especially the first ventricular sound and the blood pressure low as in the fatty heart of alcoholics. In other cases, where the blood finding is positive, as in pernicious anemia, etc., there are generally anemic murmurs over the heart and cervical vessels.

ABSENCE OF CYANOSIS IN ANEMIA

In anemic conditions there is usually no cyanosis in spite of the dyspnœa, because the blood is poor in red

blood cells and hemoglobin. Cyanosis will not even appear if there are organic affections of the muscular substance of the heart, which would otherwise lead to cyanosis. The quantitative changes of the blood are partly decisive for the absence of cyanosis, also the diminished demand for oxygen by the tissues, and in part the reduction of the carbon dioxid in consequence of the acidulation of the blood. The appearance of cyanosis, if only as a livid coloration of the lips, is therefore of increased diagnostic importance in cases of grave anemia, because it indicates considerable degeneration especially of the right ventricle, if dilatation of the cardiac cavities, negative findings in the lung, and absence of hemoglobinuria are demonstrable. Even in the gravest forms of pernicious anemia, the disturbance of circulation does not always correspond to the extent of the anatomical lesion of the cardiac muscle. The autopsy sometimes discloses friable muscular substances, while during life there were no particular indications of inefficiency of the degenerated muscle and vice versa.

Another factor may be of importance in forming an opinion on the phenomena under discussion. Lépine and Bouloud found for normal blood the time of reduction of oxyhemoglobin about twenty minutes. In anemia this time is prolonged up to about an hour. Prolonged inhalation of chloroform or ether also lengthened the time of reduction, while bacteriemia due to virulent staphylococci made no difference. In dogs which had been rendered artificially anemic, and

in anemic patients, the time of reduction was doubled and sometimes trebled. Lépine and Bouloud found in the gases from the blood of persons who had died with grave anemia demonstrable quantities of carbonmonoxid, and consider the presence of carbonmonoxid-hemoglobin one—if not the only cause, for the retarded reduction of the blood. Should these findings be verified, a new and hitherto neglected factor, aside from deficiency of oxygen, would have to be taken into account to explain many of the phenomena and the sequelæ of grave forms of anemia.

POLYCYTHEMIA

A special position among the diseases of the blood-forming organs is occupied by affections with indefinite pathogenesis, characterized clinically by polycythemia with enlarged spleen and cyanosis; dyspnœa being absent. Cyanosis of the mucous membranes and extremities forms the chief symptom of the affection. It does not depend upon the presence of reduced hemoglobin, nor is it the consequence of disturbances of circulation, but originates from the overfilling of the distended vessels with blood abnormally rich in hemoglobin. The color of the patient is not so much a blue as an intense purple. The blood examination shows a surprising increase of erythrocytes up to over ten millions to the cubic millimeter, and a genuine plethora with increased quantity of blood which explains the high blood pressure and the tendency to hemorrhage in these patients. The pathogenesis of the

symptoms in these patients is as yet unsettled, but it is probable that the nature of the affection consists in a primary or secondary involvement of the erythroblastic medullary tissue in the bone marrow leading to increased blood formation, in a similar way as leukemia represents a hyperplastic involvement of the leucoblastic myeloid tissue with increased function.

ASTHMA DYSPEPTICUM

The asthma which is occasioned by disturbances of digestion has been designated dyspeptic asthma. According to communications by Henoch, Silbermann, and the French clinicians (Barié), this is not a mechanical dilatation of the stomach by gas, nor a mechanical dyspnoea from an elevated position of the diaphragm, but a grave dyspnoea which in specially predisposed individuals sometimes occurs immediately after the ingestion of food. It is accompanied by alarming symptoms; pallor of the skin, vertigo, precordial anxiety, frequent, superficial and at times retarded, deepened respiration without obstruction to expiration, also by rapid pulse, dilatation of the heart, sometimes by gallop rhythm, cyanosis and cold extremities. As this symptom-complex is acute, the idea suggests itself that it is a reflex irritation from the stomach. To explain the mechanism of the process, various theories have been advanced: direct paralysis of the inhibitory fibers of the pneumogastric nerve and a vasomotor spasm of the arteries of the general circulation leading to temporary inefficiency of the left

heart. In other cases, the right instead of the left ventricle was held responsible by assuming that a spasm of the pulmonary vessels induced increased pressure in the pulmonary circulation with consequent inefficiency of the right ventricle. In a similar way the paroxysmal dyspnœa of gallstone colics has been explained. This explanation cannot be entirely denied, since, according to Strubell's experiments, the existence of vasomotor processes in the pulmonary circulation is very probable. Strubell, by paralysis of the pulse-retarding fibers of the pneumogastric by strophanthin and by subsequent peripheral irritation of the pneumogastric, induced a fall in the arterial pressure, reduction of pressure in the left auricle, slight increase of pressure in the pulmonary artery and considerable increase in the right auricle. He concludes therefrom that there are active vasomotors in the pulmonary vessels.

Dyspeptic asthma presents by no means a uniform symptom-complex. Cases in which there are latent cardiac affections, as arteriosclerosis or fatty heart, should, of course, be excluded, as they do not properly belong to the group of cardiac asthma which is caused by disturbance of digestion or faulty diet. Nor should cases be included where the asthma occurs immediately after excesses in drinking and smoking, as, aside from the fault in the diet, there are toxic elements in the picture. Grave dyspeptic asthma after acute digestive disturbances, especially in children and sometimes accompanied by acetonuria, should not be designated as

simple cases of dyspeptic asthma, since they belong rather to the group of toxic or toxic-infectious asthma, occasioned by abnormal fermentation in the intestines. In these cases the signs which are usually valid for the diagnosis of intestinal intoxication will be decisive, as: change in the bacterial flora of the intestine, shown by the prevalence of Gram-positive bacteria coli, which are usually anerobic, abnormal increase of indols and scatols, furthermore fermentative products of carbohydrates in the stools, the sign of intestinal putrefaction in the urine, *i. e.*, the increase of ethereal sulfuric acid in proportion to sulfuric acid and nitrogen, also the other clinical symptoms, meteorism, borborygmi, diarrhea alternating with obstipation, foul-smelling stools, coated tongue, later vomiting, and finally the successful treatment by corrected diet and disinfection of the intestines.

It is very probable that those attacks of dyspeptic asthma which occur after meals in persons with intact thoracic organs, affect neurasthenic or hysterical subjects, or even individuals with a tendency to nervous asthma, regardless of whether there is hyperchlorhydria or achlorhydria. Cases have been described in the literature, where the physiological irritation by the gastric juice was sufficient to cause an asthmatic paroxysm. Of course, it is clear that the abnormal position of the abdominal organs—gastroptosis, enteroptosis, hepatoptosis—may act as a predisposing factor by dragging upon the sensitive nerve fibers of the diaphragm.

These cases really belong to a group of disturbances of respiration, attributable to reflex asthma and consequent upon irritation of the peripheral nerves. Transmission of the irritation to the center of respiration and to the neighboring vascular centers produces paroxysmal dyspnœa and a series of vasomotor manifestations. Considerable polymorphism of the symptoms results, and this depends partly upon the peripheral source of irritation, and in part on the tract which the peripheral irritation travels. Reflex irritation of the central apparatus may induce a continuous hyperesthesia of the affected centers, so that the latter will abnormally react to irritations which emanate from other parts of the organism.

HYSTERIA AND NEURASTHENIA

The polymorphous forms of the disturbances of respiration, which occur in hysteria and neurasthenia, furnish proof of the condition described. If the irritant affects nerve fibers, irritation of which is capable of inhibiting respiration, as the superior laryngeal, the nasal branches of the trigeminus, the branches of the olfactorius, glossopharyngeus, splanchnicus and pulmonary fibers of the pneumogastric, dyspnœa with retardation and deepening of respiration ensues. In other cases where the accelerator nerves of respiration are irritated, the respiratory movement will become abnormally frequent and superficial. This respiratory neurosis, called tachypnœa, may be accompanied by tonic and clonic spasms of the inspiratory and expiratory

muscles, and by involving the vasomotor center in the medulla oblongata may produce manifestations dependent upon the latter. Inhibitory and exciting impulses, however, may be transmitted to the center of respiration not only from the periphery, but also from the cerebrum, from the motor cortical zones and the subcortical centers of respiration, *i. e.*, from the corpora quadrigemina which are connected with the center in the oblongata.

TYPE OF HYSTERICAL DYSPNŒA

These physiological processes must be taken into account in the consideration of hysterical dyspnœa and in the explanation of the multiformity of its manifestations. Nevertheless there is a definite type of hysterical dyspnœa. The hysterical forms of dyspnœa are occasioned in many cases by spasms or paralysis of the muscles of respiration—glottis and diaphragm. The clinical picture depends upon the intensity of the spasm and paralysis as well as on the localization. In other cases, the dyspnœa appears as a central respiratory neurosis without any mechanical obstruction on the part of the muscles of respiration. Diagnostic difficulties are presented by those cases where hysteria is combined with organic lesions of the lung, heart, aorta and the nervous system, as the accompanying hysterical manifestations modify and complicate the original affection.

One of the most frequent and grave hysterical affections is laryngospasm, which may vary from the

slightest rudimentary form of inspiratory sigh to the gravest type of asphyxia. This may even lead to death, while at autopsy no organic lesion of the internal organs can be discovered. Some authors (Landgraf, Lublinski) describe a hysterical tracheospasm caused by a contraction of the smooth muscular fibers of the posterior tracheal wall, which appears visible by the tracheoscope as a prominent muscular ridge underneath the mucous membrane. In a case reported by Chaput, dyspnœa and stridor disappeared after catheterizing the trachea and painting it with cocain. The provocative factor may be a laryngeal catarrh, and the affection which in itself is slight may in children produce manifestations of suffocation similar to pseudo-croup.

In other cases there is an hysterical paralysis of the posterior cricoarytenoid muscle. The inspiratory dyspnœa, the noisy respiration, which can be heard at a distance, may in these cases attain a high degree. But even in the majority of the gravest cases of dyspnœa there is neither cyanosis nor venous engorgement. Cases have certainly been described where the stormy stenotic manifestations gave cause for resorting to tracheotomy. Expiration is unimpeded and the voice is, as a rule, not changed. The laryngeal crisis of tabes, the spasms of the glottis due to foreign bodies, laryngospasm occurring in epileptics and the compression of the recurrent laryngeal in the mediastinum must be differentiated in addition to affections of the larynx.

An interesting case of hysterical dyspnœa, described

by Loubry, occurred in a man twenty-seven years of age. The severe dyspnœa was characterized by deep, forced inspiration and labored expiration, by retardation of respirations which were separated by long pauses so that there was a period of apnœa with tetanic inspiration, cold skin and anesthesia. The periods of apnœa lasted from a quarter of a minute to two minutes, after which there was a deep, noisy inspiration. The entire attack lasted about three-quarters of an hour to an hour. The periods of apnœa became more frequent and lasted longer; cyanosis increased; there was irregularity of the heart beat; tetanic contractions and opisthotonus. Upon pressure in the right iliac fossa the attack subsided, after which there was sleep, and upon awakening, normal breathing. There were, however, after the awakening, hysterogenous areas below the mamillæ, in the axilla and in the iliac fossa. The attack was repeated three times and was on each occasion overcome by pressure upon the right iliac fossa. This case resembles the pronounced dyspnœa of the pneumogastric which occurs in dogs after bilateral vagotomy, when the respirations are tetanic and followed by deep, noisy expirations and the intervals between the respirations are protracted. Respiration such as occurs in vagotomized dogs has been observed by Egger in a tabetic patient. The breathing was reduced to six respirations per minute and after several deep respirations there were intervals in the breathing up to ten seconds.

A further cause of dyspnœa in hysteria is a func-

tional disturbance of the diaphragm of the nature of a spasm and paralysis. The most frequent form is the clonic diaphragmatic spasm—*singultus hystericus*. Though the clonic spasms are frequent, the tonic spasms of the diaphragm which lead to manifestations of suffocation and to obstruction of respiration even to asphyctic symptoms are inversely rare. Fischel observed attacks of dyspnœa and cyanosis in consequence of a tonic spasm of the diaphragm on the third day of a normal confinement. Hysterical paralysis of the diaphragm is equally rare and also that form in which paralysis of the thoracic muscles must be assumed and where the respiratory movements occur exclusively by exaggerated contractions of the diaphragm. A case, described by Calvare, a twenty-five-year-old woman, showed complete immobilization of the thorax and exclusively abdominal breathing.

One of the most frequent disturbances of respiration in hysteria is hysterical tachypnœa, in which the number of respirations may be increased to sixty, one hundred or more in a minute. Breathing is superficial and irregular, of the upper thoracic type, and with slight participation or entire absence of action of the diaphragm. In spite of the stormy breathing and the enormous acceleration of respiration which is accompanied by marked gasping—a picture which Erb has compared to the panting of chased and heated dogs—the pulse is normal and the general condition not disturbed. True dyspnœa is absent, as is cyanosis. These attacks of tachypnœa last a few minutes or

longer, and disappear during sleep or by distracting the attention. Other hysterical manifestations, attacks of crying or laughing, may accompany the condition and not infrequently it is preceded by prodromal signs, as globus hystericus, dysphagia, etc. Diagnostic difficulties can arise only if hysterical tachypnoea develops in the course of organic diseases of the lung (pneumonia) or of the organs of circulation. This happened in a case of Weir-Mitchell, where a neurosis occurred after an injury to the head (with dyspnoeic manifestations).

The starting point of the hysterical disturbances of respiration is sometimes, as is well known, a disease of the genitals. Odebrecht cured a chronic spasm of the diaphragm in a young woman by curetting the affected uterine membrane, and Chrobak observed in a hysterical patient with a retroflexed uterus, grave spasms of respiration with threatening dyspnoea and cyanosis, which did not return as long as the uterus was kept in its normal position by a pessary.

DISTURBANCES OF RESPIRATION IN NEURASTHENIA

Still more complicated are disturbances of respiration in neurasthenia. Here symptoms are present which must be attributed to disturbed function of the vasomotor center. In the paroxysms of fear, respiration is usually accelerated and snoring, but the dyspnoea is more subjective. Cyanosis is entirely absent if the organs of respiration and circulation are normal. In some cases, a high position of the diaphragm is noted

in patients who swallow air during the attacks and thus distend the stomach. In these patients there may also be eructations. It is simply the frequency of states of anxiety which differentiates neurasthenic from hysterical asthma. Neurasthenic asthma interferes with the patients' sleep and even awakens them when asleep, while hysterical asthma disappears during sleep (Brügelmann.) It is impossible in these cases to give general diagnostic rules, because it is just in this field of functional neurosis that combinations are so frequent which make the pathological picture still more varied.

REFLEX ASTHMA

It has frequently been pointed out that asthma may be produced by reflex action from the various organs. The relation between nasal polypi and bronchial asthma is sufficiently familiar. There are still to be mentioned cases of asthma which occur when foreign bodies are in the ear. In a child observed by me, dyspnœa disappeared after the removal of a pea from the ear. Another case is also interesting. A patient living in the country was unsuccessfully treated by physicians for asthma. A young physician, spending his vacations in that section, remembered a clinical lecture on the subject of asthma verminosum, and was fortunate enough to think of tenia. He diagnosed the trouble correctly by examining the feces, and cured the patient of his asthma by an anthelminthic.

ORGANIC CEREBRAL LESIONS

Finally, dyspnœa may originate from organic affec-

tions affecting the innervation and motion of the respiratory apparatus. The disturbances in breathing which occur in cerebral hemorrhage, pachymeningitis and meningitis, are to be looked upon as paralyses or disturbances of co-ordination. The retraction of the paralyzed half of the thorax in hemiplegia, especially in deep inspiration, and the retarded inspiration and premature cessation of expiration which occur in other cases, prove that respiration may be influenced by the higher regions of the brain, situated above the center of respiration.

Cheyne-Stokes' phenomenon and similar intermittent disturbances of respiration are frequently found in organic cerebral lesions, hemorrhages and softening, as well as in uremic conditions toward the end of life. In basilar meningitis and especially in tubercular meningitis, there exist disturbances of co-ordination consisting in irregular movements of the respiratory muscles, chiefly the thoracic muscles and the diaphragm. Frequently, especially in the final phase of the disease, the respiration becomes irregular and accelerated, and here Cheyne-Stokes' respiratory phenomenon is observed as a premortal sign. In all these irregularities of respiration, there is, aside from the stertorous breathing, usually no true dyspnœa.

ORGANIC SPINAL LESIONS

The condition is different in direct lesions of the center of respiration as in atrophy of the nerve nuclei in bulbar paralysis. The faintness and weakness of

the voice, even complete aphony, owing to paralysis in the area of the recurrent laryngeal, as well as the dyspnœa due to paralysis of the posterior crico-arytenoid are well known. The dyspnœa may attain a high degree where there is weakness of the thoracic muscles, especially when a patient is attacked by bronchial catarrh. Paroxysms of severe dyspnœa with fright and danger of suffocation may follow interference with swallowing, in consequence of paralysis of the muscles of deglutition allowing food to enter the larynx. Effects similar to that in bulbar paralysis may also follow lesions of the bulbar nerve nuclei in other affections as amyotrophic lateral sclerosis, acute and subacute polyencephalitis, hemorrhages, foci of softening and tumors of the oblongata as well as infantile paralysis and syringomyelia. The polymorphous symptom-complex of the laryngeal crises of tabes, which are accompanied by cough, dysphagia, vertigo and dyspnœa on the one hand, and fear and sensation of suffocation on the other, belong in this category and are attributed to lesions of the nuclei of the pneumogastric and accessory nerves. In other cases there is a degenerative neuritis of the pneumogastric and recurrent laryngeal nerves, in which cases paralysis of the laryngeal muscles, and especially the posterior crico-arytenoid, may cause a picture of the most intense inspiratory dyspnœa.

Affections of the spinal centers of respiration which are the origin of the respiratory nerves may lead to dyspnœa from paralysis of the respiratory muscles.

These forms of paralysis are observed in affections of the cervical region of the cord, in caries, neoplasms, and fractures of the cervical vertebræ, in acute and chronic myelitis, in poliomyelitis and amyotrophic lateral sclerosis. In the beginning of the affection, dyspnœa may be intermittent and may have a spasmodic character like asthma, owing to irritation of the bulbo-medullary centers. These destructive affections of the spinal cord may injure directly the spinal centers of respiration, and also the tracts leading from the bulbar center of respiration to the spinal centers, thus causing dyspnœa. As the center for the phrenic nerve lies in the fourth cervical segment, it is understandable why paralysis of the diaphragm is frequently observed to occur in affections of the spinal cord and meninges which cause an injury to the third and fourth cervical roots. Aside from spondylitis, tumors, pachymeningitis and spinal hemorrhages, syphilitic meningeal affections especially lead to paralysis of the phrenic nerve. In poliomyelitis, in progressive muscular atrophy and in tabes, there may be unilateral or bilateral paresis and paralysis of the diaphragm.

NEURITIS

In the course of acute polyneuritis of toxic or infectious origin (alcohol, lead, beri-beri, diphtheria, influenza, articular rheumatism, Landry's paralysis), neuritis of the phrenic nerve, may lead to a paralysis of the diaphragm, from the extension of inflammatory processes to the phrenic nerve itself. The latter may

happen after pleuritis, pericarditis, compression of the nerves through indurative contractions in the apices of the lungs, also by the growth of neoplasms in the thoracic space, and finally in traumas as stab wounds of the neck.

In paralysis of the diaphragm there is accelerated respiration, but usually no dyspnœa, during rest. But with the least exertion, even speaking or coughing, the dyspnœa may reach an extreme degree, especially when there are simultaneous inflammatory affections of the respiratory system. On examination of the patient during quiet respiration, there will be found an enlargement of the epigastric angle and a distention of the upper half of the thorax. On deep inspiration the costal arches and epigastrium will sink in, as observed by Duchenne, while the upper half of the chest is often still further distended with the aid of the respiratory cervical muscles and atelectatic râles may be heard at the margin of the lungs. Litten's diaphragmatic phenomenon is absent and the respiratory mobility of the lower border of the lungs is not demonstrable. On the other hand, the position of the paralyzed diaphragm, even during immobility, is high, shown also in the Roentgen picture, and there is aspiration of the abdominal organs into the thorax, and a corresponding tympanitic sound at the base of the lung. Variations may be present owing to differences in degree, and as to whether the paralysis is unilateral or bilateral.

Paralysis and atrophy of the intercostal muscles which occur frequently after polyneuritis, in rare cases

also in poliomyelitis, display a behavior opposite to that of diaphragmatic paralysis. In deep inspiration, distention of the upper half of the thorax is absent, while the action of the diaphragm causes a bulging of the epigastrium and a retraction of the base of the thorax.

The significance of the pneumogastric nerve for the production of dyspnœa, frequently accompanied by cardiac manifestations, has been repeatedly mentioned. It only remains now to refer once more to the degenerative and neuritic affections of the pneumogastric nuclei and trunk, which are partly primary and in part secondary.

Insufficiency of the diaphragmatic function may cause dyspnœa, possibly also cyanosis, in the way already explained when speaking of trichinosis, in primary affections of the respiratory muscles, as myositis of the diaphragm or inflammatory processes, which originate from neighboring organs, as diaphragmatic pleuritis, also not infrequently from suppurative processes in the abdominal area, as subphrenic abscesses consequent upon *ulcus ventriculi*, perityphlitis, exulcerative carcinoma of the stomach or carcinomatous infiltration of the lymphatic vessels of the diaphragm.

MYOSITIS

In acute hemorrhagic polymyositis, in which the entire transversely striated musculature and the subcutaneous tissue may be involved, simultaneous and anal-

ogous affections of the muscular substance of the heart produce a pathological picture in which, after preceding tachycardia or arrhythmia, severe dyspnœa and fear with general muscular spasms, form the dominating symptoms.

IX

THERAPY OF DYSPNŒA

The therapy of the various forms of dyspnœa will depend on the nature of the original affection. In the cardiac forms of dyspnœa occasioned by valvular defects, digitalis will be indicated in the vast majority of cases, if cyanosis predominates. When there is simultaneous anemia with hard, accelerated pulse, as occurs in insufficiency of the aortic valves and especially in the arteriosclerotic form, with exacerbations of dyspnœa at night, digitalis will avail nothing or even make the condition worse. In such cases a milk diet, in others potassium iodid, and finally preparations of bromid or morphin are indicated. In dyspnœa, caused by pulmonary edema with active hyperemia of the lungs, good service will sometimes be rendered by local bleeding, dry or wet cupping, moist packs to the chest, counter irritant remedies like mustard, or local mustard baths of hands or feet.

Coincident renal insufficiency requires caution in the administration of digitalis. In disturbances of compensation and in degeneration of the cardiac muscle, combinations of small doses of digitalis with caffein, camphor, ether, alcohol, and in some cases spartein, are many times of benefit. In other cases, where there is rapidly developing cyanosis with dilatation of the

right ventricle, bleeding may remove a threatening edema of the lungs, especially if the symptoms of cerebral and pulmonary congestion are not benefited by the cardiac remedies. The same is true of dyspnœa with grave cyanosis in deformities of the thorax.

In effusions into the serous membranes, diuretic therapy may be employed (diuretin, agurin, theocin), and according to circumstances all of the vegetable diuretics should be tried, as they exhibit considerable differences in individual cases. One patient may react to scilla, another to equisetum or ononis; even obsolete or lay vegetable remedies may be of advantage.

Narcotics such as opium and its derivatives probably cannot be entirely dispensed with where dyspnœa is caused by irritation of the cardiac or periaortic plexus, or of the pulmonary fibers of the pneumogastric, as in dyspnœa of bulbar origin consequent upon ischemia of the oblongata, also in some forms accompanied by attacks of stenocardia. Nitrits, nitroglycerin, erythroltetranitrat, spiritus ætheris nitrosi, amylnitrit alone or in combination with strophanthus or even digitalis, may in a similar way relieve the patient. Some patients are quieted by oxycamphor.

In the cardiac dyspnœa of old persons with weak hearts, also in the dyspnœa of diseases of the blood, arsenic is sometimes effective, either alone or in combination with digitalis and strophanthus. The dyspnœa of fatty heart in obese people should be treated by dietary measures.

In bronchial asthma, treatment with stramonium,

iodin preparations and morphin is sufficiently known. The treatment of emphysema of the lungs has already been described.

Dyspnœa in pneumonia requires individual treatment according to indications. Where there is much oppression with cyanosis, dilatation of the cervical veins, abundant bloody expectoration, bleeding may sometimes be indicated in strong individuals; in other cases, cold packs; while in still others, amylnitrit may do well.

The treatment of dyspnœa in phthisis depends upon its cause. Indications will differ in pneumothorax, febrile dyspnœa and in the simultaneous involvement of the heart and pericardium.

In bronchitis with scant expectoration, expectorants like ipecac, senega and antimony may become necessary. In diphtheria antitoxin treatment, emetics, intubation or tracheotomy, in edema of the glottis cupping, leeches to the neck, drastic purgation or surgical interference.

For the treatment of renal dyspnœa, no general rules can be established. There are cases in which an exclusive milk diet alleviates the symptoms, in others theobromin preparations, and in still others, diuretics and drastic remedies, ether in large doses, even bleeding, morphin injections, and in some cases lumbar puncture. In diabetic acid dyspnœa, large doses of soda bicarbonat should be tried, also intravenous injections of the same, which, however, are nearly always useless.

Inhalations of oxygen in the treatment of dyspnœic conditions in various affections enjoy a certain reputation. In carbonmonoxid intoxications, in which the tissues are insufficiently supplied with oxygen in consequence of the formation of carbonmonoxidhemoglobin, also in intoxications with agents which form methemoglobin, oxygen therapy both in the form of inhalations and by rectal administration is advisable and theoretically well founded.

A further indication for oxygen is morphin poisoning. We have, ourselves, seen a grave case of morphin poisoning which was saved by tracheotomy and artificial respiration, with pure oxygen administered through the tracheal canula.

In other forms of dyspnœa of mechanical origin, as in stenosis of the upper air tract, emphysema, cardiac asthma, dyspnœa in phthisis, also in pneumonia, in affections of the blood, as pernicious anæmia and leukemia, there are often cases in which inhalations of oxygen give the patients at least some alleviation of the subjective difficulty in breathing.

DISORDERS OF METABOLISM AND NUTRITION

A Series of Monographs by **Prof. Dr. CARL von NOORDEN**

Physician-in-Chief to the City Hospital, Frankfort-on-Main

Authorized American Edition Translated under the Direction of **BOARDMAN REED, M.D., PHILADELPHIA.**

IT is due to the disorders of metabolism and nutrition that degenerative changes cut short the activities of so many men and women in middle life,—that, in these latter days, senility and death itself come prematurely to a very large proportion of mankind. Such disorders constitute the bane of our modern civilization. They have been in some measure also a reproach to the science and art of medicine, since until very recently they have not been studied with a thoroughness commensurate with their importance.

I. OBESITY, THE INDICATIONS FOR REDUCTION CURES.—In this volume the disease is considered in a manner which is at once scientific and practical; based upon exhaustive experiments and bedside observations carried on under the direction of the author.

Cloth, 8vo, 60 pages, 50 cents.

II. NEPHRITIS.—The author's handling of this subject is varied and original, and in various respects he has established for the treatment of the different forms of Bright's disease, rules founded upon a critical, scientific study of numerous cases instead of the familiar directions handed down from an earlier period.

Cloth, 8vo, 112 pages, \$1.00.

III. COLITIS.—The author covers the complex subject of Membranous Catarrh of the Intestines (*Colica Mucosa*), in a manner which is well-nigh exhaustive and also most convincing since he is able to report a remarkably large proportion of cures obtained by the method which he recommends.

Cloth, 8vo, 64 pages, 50 cents.

IV. THE ACID AUTOINTOXICATIONS.—These studies into the derangements of metabolism, which result in an overproduction of acid, concern the clinician very nearly. They are in a field which has been hitherto too little explored.

Cloth, 8vo, 80 pages, 50 cents.

V. SALINE THERAPY.—The author here decides many mooted questions concerning the influence of the sodium chloride waters on the digestion, as well as in gout, diabetes and other diseases of nutrition.

Cloth, 8vo, 96 pages, 75 cents.

VI. DRINK RESTRICTION. (Thirst Cures).—A most instructive deliverance upon a subject of the highest practical importance.

Cloth, 8vo, 90 pages, 75 cents.

VII. DIABETES MELLITUS, A Series of Lectures delivered before the University and Bellevue Hospital Medical College, New York. Oct., 1905.

Cloth, 8vo, 212 pages, \$1.50.

VIII. GOUT. In Press.

E. B. TREAT & CO., Publishers, Nos. 241-243 West 23d Street
NEW YORK

DISEASES OF THE Stomach and Intestines

As well as the Allied and Resultant Conditions, with
Modern Methods of Diagnosis and Treatment

By **BOARDMAN REED, M.D.**

THE AUTHOR has had large experience in this special field of medicine, supplemented by knowledge acquired during twenty years spent in a busy general practice among chronic invalids in Atlantic City, postgraduate work both in this country and abroad, and teaching in this special department, and is eminently qualified for the task he has so successfully completed.

THE VOLUME covers so comprehensively the etiology, pathology, symptomatology, diagnosis and treatment of the various diseases in question that it stands as the only thoroughly up-to-date single volume work on the diseases of the stomach and intestines. The instruction is so plain and simple that every general practitioner, as well specialists in other lines, will find it a real help in the countless puzzling cases complicated with, or wholly dependent upon, derangements in the digestive system.

SPECIAL FEATURES are "The Gastrointestinal Clinic" in which the diagnosis and treatment of all known diseases of the tract are separately considered; a very complete "Symptomatic Guide to Diagnosis"; an account of the relations of gastrointestinal diseases to numerous other affections, such as Neurasthenia, Insomnia, Heart Disease, Kidney Disease, etc., and a comprehensive account of the diagnosis and treatment of Diseases of the Rectum and Anus contributed by Dr. COLLIER F. MARTIN, the well-known specialist.

"The author is clear and sound in his teachings, simplifies conditions as far as possible, and gives a good practical working knowledge, such as he has gathered from a large experience in this special field."—*Medical Record*.

"The need of a modern treatise on this subject is evident from the limited literature and increasing demand for the same. Not only general practitioners, but also specialists in other lines, will find it of great value."—*American Medicine*.

Complete in One Large Octavo Volume; 1024 pages.

Profusely Illustrated. Half Morocco, \$6.00. Cloth, \$5.00

Sent Post or Express Paid on receipt of Price. Circulars upon request

E. B. TREAT & CO., Publishers
241-243 West Twenty-Third Street, New York

INDICATIONS FOR OPERATION

—IN—

Diseases of the Internal Organs

By PROF. HERMANN SCHLESINGER, Vienna

AUTHORIZED ENGLISH TRANSLATION

By K. W. MONSARRAT, F.R.C.S. Ed., Surgeon Northern Hospital, Liverpool

From the Author's Preface—"I have written essentially for the practitioner. I have, therefore, avoided all prolix discussion, and confined myself to the consideration of the questions indicated by the special design of the work. On the advice of colleagues whom I have consulted, I have included in each chapter some remarks on etiology, pathological anatomy, clinical course, diagnosis and differential diagnosis, with a view to enabling the practitioner to quickly obtain a general grasp of the condition under consideration. These paragraphs, are, however, subordinate to the main purpose of my undertaking—that of enabling medical men who are not in hospital practice to arrive at an independent opinion on the advisability of operation in a large majority of cases of internal lesion. This is not a mere compilation. Many years' work in hospital and private practice have given me the opportunity of acquiring a large experience in the questions here discussed."

American Journal of Surgery, New York, says: "We do not recall that this subject has ever been touched upon in a separate volume before, or even adequately in volumes devoted to collective discussions. This work may therefore be said to fill a definite want. It is novel in another respect, namely, that a presumably surgical subject is discussed by a medical man. This feature, we believe, adds greatly to the value of the book, inasmuch as it introduces a point of view that is only too frequently wanting."

Medical Standard, Chicago, says: "A most timely and distinctively purposeful work. Of all the problems which the wide-sweeping activities of operative surgery have imposed upon the general practitioner, the question of when to operate and when not to, is the most difficult and delicate. A guide to the solution of this hydra-headed problem, therefore, and from an authority so universally recognized as Dr. Schlesinger, cannot fail to be peculiarly welcome. All will do well to read, mark, learn, and inwardly digest."

One Volume, 8vo, (8¾ x 5½ inches), 514 pages, cloth, net, \$3.00

E. B. TREAT & CO., 241-243 W. 23d St., New York

MEDICAL AND SURGICAL USES OF ELECTRICITY

INCLUDING
**X-Ray and Vibratory Therapeutics, the Finsen Light and
High Frequency Currents**

BY
A. D. ROCKWELL, A.M., M.D.

*Formerly Professor of Electro-Therapeutics in the N. Y. Post-Graduate Medical School and
Hospital; Fellow of the New York Academy of Medicine; Member of the American
Academy of Medicine; Member of the New York Neurological
Society; Formerly Electro-Therapeutist to the Woman's
Hospital in the State of New York, etc.*

Electricity in its relation to medical science, as well as to commerce, has made wonderful progress in recent years. The recognized pioneer work in America was the treatise on "The Medical and Surgical Uses of Electricity," by Beard and Rockwell, of which eight successive editions were issued. In 1896 the stereotype plates were destroyed and the work rewritten by Dr. Rockwell, and issued from an entirely new set of plates. To keep abreast of the marvelous progress in this department of Medicine, a New Edition is again offered to the profession. The Roentgen X-Ray in its relation to Diagnosis and Therapeutics has been entirely rewritten. Other additions include the subject of the Actinic Rays of light (Finsen's), High Frequency Currents and Vibratory Therapeutics. In the body of the book much that has served its purpose has been discarded and new and up-to-date material substituted. It is confidently believed that in its pages will be found a comprehensive and accurate survey of this fascinating and growing field of research.

Medical Record, New York, says: "For twenty years and more the work of Beard and Rockwell has been the leading authority in this country on the subject which it treats. They were the pioneers in the field of electro-therapeutics and enunciated ideas and methods which have stood the test of time. . .

The work plainly sets forth all the fundamental principles of electricity, and in its relation to disease is clear in detail and cannot fail to greatly aid all who are interested in this department of medical science."

Medical News, Philadelphia, says: "The methods of application for therapeutic purposes are given in detail and with such clearness that the general practitioner will find the book a useful and practical guide."

Medical Journal, New York, says: "The book is much changed from the earlier editions, being in accord with the advance in our knowledge of the applications of electricity. . . . The work is valuable and will clear up many points which may be shadowy in the mind of the general practitioner."

**New, Revised and Enlarged Edition.
Royal Octavo, 692 pages; Illustrated.**

Cloth, \$5.00 net.

Half Morocco, \$6.00, net.

E. B. TREAT & CO., Publishers

241-243 West 23d Street, New York

